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SYMPOSIUM
ON
ENVIRONMENTAL
LEAD
CONTAMINATION

Sponsored by the Public Health Service
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WHY A SYMPOSIUM ON ENVIRONMENTAL LEAD CONTAMINATION

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Washington, D.C.

It is a pleasure for me personally, and on behalf of the Surgeon General, to welcome you to this Symposium on Environmental Lead Contamination.

During the next three days, you will be concentrating your attention on a single important facet of the complex and compelling problem of environmental health, the hazard imposed by lead in the environment. You will be examining the many ways by which lead enters the environment, discussing the effects of lead on human health, and exploring measures which may be needed to minimize these effects. We in the Public Health Service are deeply grateful for your assistance in this undertaking.

This Symposium is one of many manifestations of a heightened national awareness that our understanding of environmental hazards is far from adequate. As a nation we are recognizing that subtle but serious health implications are often among the by-products of our high levels of technological achievement and economic prosperity. This recognition is a necessary condition for progress. Symposia like this one are essential steps toward the broader goal of creating and maintaining healthful surroundings for man.

Across the entire range of environmental health problems we are making a rather belated start. Our record of gauging in advance the potential health risks associated with technological change is, I am afraid, a poor one. All too often government and industry have failed to keep abreast of the accelerating pace of technological changes which have profound effects on the health and welfare of all Americans.

But now we are on our way. Major efforts are being launched to clean up tens of thousands of miles of polluted waterways, to clean the air which has become a hazard to public health and welfare in thousands of communities. In addition, we need to accelerate our efforts to understand the health implications of those chemical and radiological substances to which man is exposed, not through air and water alone, but through air, water, food, and an array of other environmental factors.

Your examination in depth of lead contamination represents one important means for achieving this understanding. With respect to lead, there is a sizable base of scientific knowledge of its physiologic and toxic effects, much of it derived from studies of occupational exposure. But we must be able to assess more adequately the role of lead as an environmental contaminant to which all people, regardless of their occupations, are exposed.

The pace of technological change is so swift that what we knew yesterday about potential health risks may not suffice today, and certainly will not tomorrow. In a nation where technology is advancing so rapidly, we cannot afford to be backward. The price is too high. It is my sincere hope that those of you in this room will provide for all of us--government, industry, and the public to whom we are ultimately responsible-- an improved basis upon which we can make certain that we do not now, or in the future, take undue risks from lead in the environment.

We have made a sincere effort to bring together persons representing the widest possible range of informed scientific opinion on the subject of lead. We are seeking knowledge of the nature and extent of environmental lead sufficient to enable all of us in *government and industry to take whatever actions may be needed in the public interest. We begin these discussions at a time when there are important differences of opinion concerning the magnitude of the risk to which the public is now being subjected by the presence of lead in the environment. Some maintain that a large segment of the population is already perilously close to the threshold of lead toxicity as a result of environmental exposure; others take an almost diametrically opposed position. It is my hope that in the next three days you can move toward an objective definition of the role of environmental lead as it affects human health and welfare.*

In my view, vigorous disagreement is a most healthy sign. It stimulates investigation and discussion. It is insurance against neglect. The fact that you have come together here indicates that all of us have learned something from our mistakes of the past, that we are no longer willing to wait in dealing with environmental hazards. I look forward to the outcome of this symposium, for I am certain that you have an opportunity to help government, industry, and the people of this country to deal effectively with the problem of environmental lead.

* * * * *

PANEL: MAJOR SOURCES OF LEAD POLLUTION

Chairman: R. J. M. Horton
Co-Chairman: J. H. Ludwig

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ESTIMATION OF SOURCES OF ATMOSPHERIC LEAD AND MEASURED ATMOSPHERIC LEAD LEVELS

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This is one of a series of four presentations to summarize information on man's exposure to lead. This paper reviews literature on sources and atmospheric lead levels, and much of what is said here is taken from the recently published "Survey of Lead in the Atmosphere of Three Urban Communities." (1)

Total lead consumption in the U.S. has remained more or less steady since 1941 (Figure 1); however, individual uses have changed over the years. For example, in 1943 the principal use was red lead and litharge (18.5%) for metal protective paints. In 1953 the principal uses were for storage batteries (30.5%) and tetraethyl lead (13.5%). A further increase is shown for these two uses in 1963 (Table 1), which together accounted for 54 percent of lead used.

Lead is used in a wide variety of activities and 23 additional classifications were required in the "1963 Minerals Yearbook" (2) to account for the remaining 46 percent of the lead used in this country. In addition to the widespread industrial use, lead is widely distributed in nature in rocks, soils, water, and vegetation. Consequently, there are many potential sources for atmospheric emission of lead.

SOURCES OF LEAD EMISSION

Emissions can result from many industrial processes in which lead is used. Concentrations of lead in in-plant air have been reported to vary from less than 1 to as many as 11 mg/m^3 in insecticide manufacture, up to 38 mg/m^3 in storage battery manufacture, and 49 mg/m^3 in paint manufacture. (1) In one study of storage battery manufacture, atmospheric concentrations of lead were determined in the outdoor air. Samples taken upwind from the plant were found to have about 9 μg Pb/m^3 of air, and samples taken downwind contained as much as 90 $\mu g/m^3$. (3) Obviously, this activity increased the atmospheric lead levels at least near the plant. The data as presented were insufficient to quantitate the plant's overall contribution to atmospheric lead levels.

Lead emissions can occur when lead-bearing materials are burned. This introduces the possibility of lead emissions from municipal incineration, burning dumps, and burning waste materials associated with building demolition. For the most part data pertaining to these sources are lacking. However, some samples were taken of effluent from subterranean burning in a New Orleans dump. Two of these samples were analyzed for lead, and lead concentrations of 12 and 18 $\mu g/m^3$ were found.

Another possible source of atmospheric lead is that from the re-entrainment into the air of lead-bearing soils. This could be natural re-entrainment such as wind-blown dust, or it could be from dusty out-door operations such as heavy road traffic.

The average lead content of the earth's crust has been estimated at 16 ppm, and higher values have been reported in urban and industrial areas. Cholak found up to 360 ppm of lead in old residential soils. (4) In New York City 2650 ppm has been found in street sweepings. (5) In mining and certain industrial areas much higher values have been cited. (6)

If a lead-bearing soil were resuspended and collected as an atmospheric sample, it would take 10,000 ppm of lead to amount to 1 percent of the sample weight. By way of comparison the three-city study shows that the average content of lead in particulate matter air samples averaged 1.7 percent in Cincinnati, 1.5 percent in Philadelphia, and 2.3 percent in Los Angeles. Thus, it would appear that the fraction of lead in urban soil is much less than the fraction of lead in airborne particulates. Consequently, resuspension of urban soils is a minor source of atmospheric lead pollution.

Lead can be emitted to the atmosphere during the combustion of lead-bearing fuels. These are principally gasoline with lead additives and coal containing lead and other impurities. I could not find any literature citations that suggest lead is emitted from combustion of fuels other than these.

What are the relative contributions of these various sources to the prevailing atmospheric lead levels? Emission inventories have been made in Los Angeles, Detroit, Cincinnati, Nashville, Louisville, Erie County, New York, and elsewhere. Although these inventories deal specifically with gaseous pollutants, they deal only generally with particulate pollutants. That is, the constituents such as lead in the particulates are not normally identified.

One means of estimating the relative quantity of lead emitted when coal and gasoline are burned is to use one of the area-wide emission inventories as a point of departure, then apply whatever factors are available, and from this develop an estimate for each source. This approach was used on the recently completed emission inventory for Cincinnati and neighboring counties. (7) This area includes Hamilton County, Ohio, and Boone, Kenton, and Campbell Counties in Kentucky. The population of the area is 1,120,000.

In this area about 12,400 tons of coal are burned per day. Cholak (4) indicates that 23 samples of Cincinnati coal had an average lead content of 11 ppm. If all of the lead in the coal were emitted as fine particulate, the daily lead emission from this source would be about 270 pounds. Cuffe (8) measured trace metals, including lead, ahead of and behind air cleaners at six power plants, however, and noted that "The average collector efficiencies of trace metals for any of the six power plants were nearly the same as the fly-ash collection efficiencies..." If a modest fly-ash collection efficiency of 80 percent is applied to coal burned in the Cincinnati area, the resulting lead emission on this basis would be approximately 54 pounds per day.

About 930,000 gallons of gasoline are burned daily in this area; based on the 1962 national average of 2 grams of lead per gallon of gasoline, one can estimate that about 4100 pounds of lead are used daily. Hirschler (9) has shown that 70 to 80 percent of the lead in automotive fuels is eventually discharged from the exhaust, but less than this is discharged during city driving. He also indicated that from 50 to 75 percent of the lead discharged is of a particle size that might remain airborne. If an estimated 70 percent of the lead is exhausted in the combination of city and expressway driving that prevails in the Cincinnati area, and 60 percent of it is small enough to remain airborne, then daily atmospheric lead emission from leaded gasoline in the Cincinnati area would be estimated at 1700 pounds.

Measured Atmospheric Lead Levels

Atmospheric lead levels have been much more extensively measured than have been emissions from the various sources. The "Survey of Lead in the Atmosphere of Three

Urban Communities," (1) reports values for Cincinnati, Los Angeles, and Philadelphia, and furnishes examples of urban atmospheric lead levels.

Values found in the three-city study are summarized in Table 2.

The annual average atmospheric lead values ranged from about 1 to 3 $\mu g/m^3$ depending on whether the station was on the edge of town or more nearly in the center of town. There was a tendency for slighly higher values during the fall and winter seasons, and on a daily basis there was a tendency for highest values to occur between 0700 and 1100. The samplers at fixed stations in this study were placed to reflect area-wide pollution levels.

Table 3 represents a compilation of data from various sources. It records a series of mean atmospheric lead concentration for particular sets of samples and identifies the sampling locations. The table is arranged in increasing order of atmospheric lead values, and ranges from 0.3 $\mu g/m^3$ of lead in suburban National Air Sampling Network stations to 44.5 $\mu g/m^3$ in Boston's Sumner Tunnel.

Sampling locations representing the general environment give average results ranging from 0.3 to 2.5 $\mu g/m^3$; however, samples taken in the vicinity of auto traffic give substantially greater values, and the particular values appear to depend on traffic density and proximity of sampler to the traffic. We see, for example, the annual average lead value at four Cincinnati sampling stations was 1.4 $\mu g/m^3$; however, a driver parked on a busy residental street would experience 4.7; on a commuter route during midday, 9.1; on a commuter route during rush hour, 14.2; and parked at a downtown curb during Christmas rush, 21.4.

Summary and Discussion

There are many possible sources of atmospheric emission of lead. However, there are only very limited data regarding the quantity of these various emissions. It would appear that:

1. Industrial activity using lead is a source of pollution, but emission data are lacking.
2. Burning of waste materials containing lead is a source of pollution, but emission data are minimal.
3. Re-entrainment into the air of lead-bearing soils is a source of pollution, but for urban soils this is probably a minor contribution.
4. Coal combustion is a source of pollution, and for the Cincinnati Metropolitan Area amounts to between 54 and 270 pounds of lead per day, depending on the amount of lead retained in the ash or air cleaning equipment.
5. Combustion of leaded gasoline is a source of pollution, and for the Cincinnati area amounts to about 1,700 pounds per day.
6. General urban air pollution levels for lead may run from about 1 to 3 $\mu g/m^3$, but atmospheric measurements taken near automobile traffic may run up to about 40 $\mu g/m^3$, depending on proximity to traffic and traffic density.
7. Based on available data it appears that the principal source of atmospheric lead in urban areas is that resulting from combustion of leaded gasoline.

TABLE 1

LEAD CONSUMPTION IN THE UNITED STATES (2)
DURING 1963

	1000 Short Tons	Percent
Storage batteries	439	37.5
Gasoline antiknock additives	193	16.5
Caulking lead	76	7
Red lead pigment	71	6
Solder	68	6
Cable covering	58	5
Ammunition	50	4
Other (18 classifications)	208	18
	1,163	100

TABLE 2

CONCENTRATION OF LEAD IN ATMOSPHERE (1)
($\mu g/m^3$)

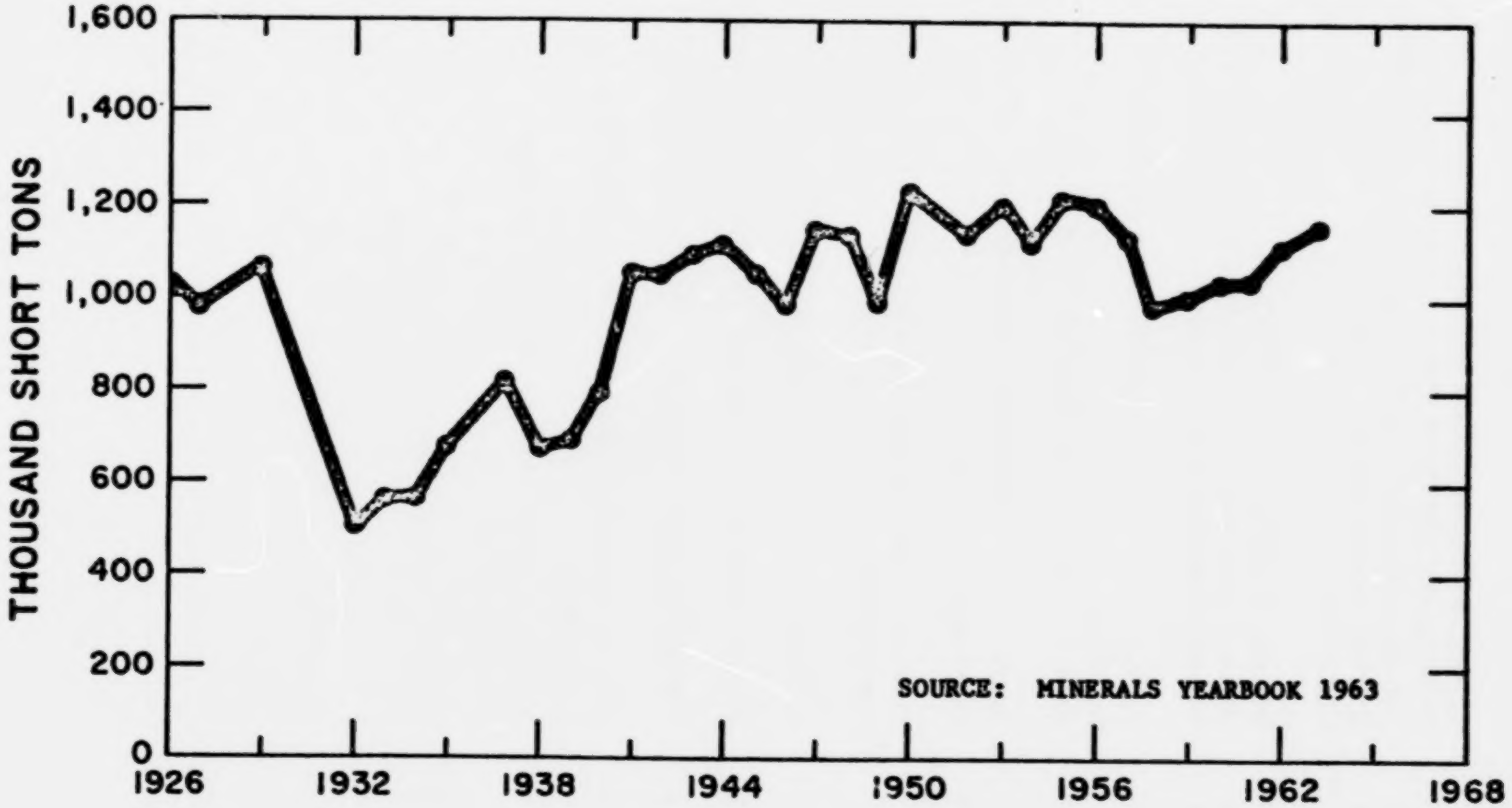
Circumstance	Cincinnati	Los Angeles	Philadelphia
Annual Average Value:			
downtown	2	3	3
outlying area	1	2	1
all stations	1.4	2.5	1.6
Seasonal Distribution (all stations)			
summer	1.3	1.9	1.4
fall	1.7	2.8	1.9
winter	1.3	3.1	1.9
spring	1.3	2.1	1.4
Diurnal Distribution (annual - all stations stated as a fraction of annual mean)			
2300 - 0300	1.3	1.0	0.9
0300 - 0700	1.1	1.1	0.8
0700 - 1100	1.4	1.2	1.4
1100 - 1500	0.8	0.7	0.8
1500 - 1900	0.9	0.8	1.1
1900 - 2300	1.0	1.1	1.1

TABLE 3

CONCENTRATION OF LEAD IN ATMOSPHERE WITH RESPECT TO SAMPLING LOCATION

Mean lead $\mu g/m^3$	No. of samples	Sampling location	Source
0.3	149	NASN surburban stations, 1954-1956	10
0.5	819	NASN urban stations, 1957-1960	11
0.8	651	NASN urban stations, 1959-1962	12
1.2	5	Parked in open area about 200 yds. from road - Detroit	13
1.4	2007	NASN urban stations, 1953-1957	10
1.4	600	Annual av. 4 stations, Cincinnati	1
1.6	1200	Annual av. 8 stations, Philadelphia	1
1.9	5	Parked at curb, 220 cars/hr., Cincinnati	
2.0	4	Parked near outlying thoroughfares - Cincinnati	14
2.5	1200	Annual av. 8 stations, Los Angeles	1
2.8	5	Parked near downtown traffic - Detroit	13
3.1	13	Rural cruising at 30-40 mph. - Cincinnati	1
4.0	16	Parked 500 feet from heavy traffic	14
4.3	4	Rural stop and go behind another car - Cincinnati	1
4.6	321	At filling station, total of organic and inorganic - Los Angeles	14
4.6	15	Parked at curb, 500 cars/hr. - Cincinnati	1
4.7	15	Parked on busy residential street - Cincinnati	14
4.8	13	Parked at curb, 820 cars/hr. - Cincinnati	1
6.7	266	At filling station, close to gas tanks, total of organic and inorganic - San Francisco	14
7.6	12	Parked at curb, 2150 cars/hr. - Cincinnati	1
7.7	12	Parked by heavy traffic - Cincinnati	14
8.2	10	Parked near freeway interchange - Detroit	13
9.1	8	Driving commuter routes, midday - Cincinnati	1
9.4	4	Driving, downtown traffic, Saturday - Los Angeles	1
10.5	4	Driving, downtown traffic, weekday, midday - Los Angeles	1
11.5	3	Driving, downtown during afternoon rush - Cincinnati	1
11.9	6	Driving, downtown traffic, midday - Pasadena	1

Mean lead $\mu g/m^3$	No. of samples	Sampling location	Source
12.4		Driving, downtown traffic, Saturday - Pasadena	1
13.1	10	Driving, on commercial streets - Cincinnati	14
14.2	20	Driving, commuter routes, morning rush - Cincinnati	1
14.3	35	Driving, streets loaded heavily, 2150 cars/hr. Cincinnati	1
15.1	25	Driving, in heavy traffic - Cincinnati	14
15.2	8	Driving, commuter routes, afternoon rush - Cincinnati	1
15.3	4	Driving, downtown traffic, weekday - Los Angeles	1
18.4	7	Parked along freeway, weekday, afternoon rush - Los Angeles	1
19.9	4	Parked along freeway, Saturday - Los Angeles	1
21.4	4	Parked at curb, downtown, Christmas rush - Cincinnati	1
21.5	39	Driving, freeway traffic, weekday, midday - Los Angeles	1
22.2	34	Driving, freeway traffic, weekday, afternoon rush - Los Angeles	1
23.6	6	Driving, downtown, weekday, morning rush - Los Angeles	1
24.1	7	Parked along freeway, weekday, midday - Los Angeles	1
25.3	32	Driving, freeway traffic, Saturday - Los Angeles	1
29.3	35	Driving, freeway traffic, weekday, morning rush - Los Angeles	1
38.0	7	Parked on major freeway, weekday, morning rush, downtown - Los Angeles	1
44.5	7	Sumner Tunnel, outlet air - Boston	15



SOURCE: MINERALS YEARBOOK 1963

Figure I. LEAD CONSUMPTION IN THE UNITED STATES, 1926-1963.

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THE DIET AS A SOURCE OF LEAD POLLUTION

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Exclusive of individuals accidentally poisoned or occupationally exposed, it is generally agreed that the diet is the major source of lead exposure and contributes about 90 percent of the total intake. Techniques used to estimate dietary exposure include the direct determination of lead in whole diets and the calculation of lead intake from either the lead content of individual items that comprise a diet or the amount of lead excreted by the individual. The latter technique is based on knowledge that less than 10 percent of the lead ingested is absorbed from food in the alimentary tract and that lead is excreted from the body largely through the feces and to a lesser extent in the urine. These relationships are relatively constant and, therefore, the amount excreted from the body by either route can be used as a basis of estimating lead intake. These observations were made by Kehoe better than 20 years ago. Using this technique, at that time he also determined that the average lead intake for people in the United States and in Europe was about the same. Samples obtained from Mexico suggested a lower level of intake. Most of the values fell between 0.1 and 2.0 mg lead per day with an average value of 0.3 mg per day. Monier-Williams, for individuals in England, estimated the average daily intake from food to be 0.22 mg lead per day. About 20 years later (1961) Schroeder and Balassa estimated the average daily intake, from the analysis of a wide variety of food commodities, to be 0.29 mg lead per day. Considering the time span and geographical area covered, the consistency of results is remarkable.

Since the diet is composed of a wide variety of individual items, it is relevant to consider the lead content of various foods. In comparing the various analyses of Kehoe, Mexico and U.S.-1940 (1), Bagchi, India-1940 (5), and Schroeder and Balassa, U.S.-1961 (4), it was noted that no one food or group of foods was either a large or constant contributor of lead to man. To illustrate, it was noted that Schroeder and Balassa (1961) found 2.5 ppm lead in lobster while Bagchi (1960) observed 0.08 ppm. Kehoe found 2 to 4 ppm for beets and carrots while Bagchi reported 0.21 ppm. The extremes reported by all three investigators ranged from 7.5 ppm for one item to 0 ppm for several items, with an average value of about 0.2 ppm. Assuming that man consumes daily about 2 kg of food containing 0.2 ppm lead, his average daily intake would be 0.4 mg per day which is consistent with the reported values.

Of particular interest to us is the amount of lead in milk. Milk is an important source of food, particularly for children, and it and its products are produced and used throughout the country. A point of interest in speaking of milk was an experiment conducted by Marshall et al (10) in which eight Jersey cows were fed up to 12.95 mg lead per 100 pounds of body weight per day for 126 days. All the milk samples analyzed, including those from the control cows, had a lead concentration less than 0.05 mg/l. On the other hand, Kehoe (10) reported two surviving cows from a dairy herd which had ingested lethal doses of lead oxide showed twelve days later 2.26 and 0.15 ppm in their milk and 122 days later showed about 0.03 ppm in the milk. These results suggested that the level of lead in milk is a function of the body burden of the element rather than that in the daily diet. If this be so, then the lead concentration in milk should reflect an integrated exposure of the cow to lead through her food chain. Thus, this would serve as a useful indicator for significant changes in the environmental exposure of man to lead. Kasahara et al (1936) gave values of 0.02 to 0.04 ppm for United States milk samples and 0.01 to 0.06 ppm for Japanese milk. Shephard et al (1942) found the average lead content of 41 market milk samples as 0.14 ppm and considered that the main source of lead in milk was the 10 gallon bulk milk can. Murthy and Rhea, in our laboratories, recently analyzed 37 milk samples collected in glass in the Cincinnati area and found the level to be 0.04 ± 0.017 ppm. Also analyzed at the same time were 35 market milk samples from northern and eastern portions of the United States in which they found 0.05 ± 0.025 ppm.

Although a critical comparison of the values of Murthy and Rhea with earlier investigators is not possible, there is a suggestion in these data that the level of lead in milk is slightly higher today than 20 to 30 years ago.

There are many factors which may influence the concentration of lead in food. Among the more important of these are the concentration of lead in the growing environment of plants and food producing animals, (the uptake, excretion, retention, and distribution of lead by the organism), contamination with lead pollutants through aerial fallout, use of lead compounds in agriculture, and direct contamination during processing, storage, and final preparation.

Borisova analyzed the lead concentration in the soils of two different areas, the Republic of Tartar and the Republic of Armenia. The soil in the Tartar area had up to 30 ppm while that in the Armenia area had up to 2000 ppm. He then analyzed the crops grown on each of the soils and found that the crops grown in the soil with the high lead content had 2 to 10 times the amount of lead compared to the crops grown in the soil with the lower lead concentration. Warren and Delavault (6) demonstrated that lead resulting from pollution by automobile exhaust and industrial uses can contaminate the plant in a manner not unlike that of growing in a lead-rich soil.

The use of lead containing insecticides has had a long and honorable history in the development of modern agriculture. Prior to the development of the chlorinated hydrocarbon and organic phosphate insecticides, lead arsenate was used extensively, particularly on apples, cabbage, and celery. Today it is restricted to specialized problems, such as checking the cherry fruit fly, and its production has dropped from 30 million pounds in 1956 to less than 9 million in 1963.

In times past, direct contamination of foods with lead has been (if recognized) of great concern for there are many cases in the historical literature of plumbism associated with the ingestion of food and drink exposed to lead during processing, storage, and final preparation. In recent years, particularly since World War II, the equipment used in food industries has been constructed almost exclusively from stainless steel with welded joints which contain no lead. Similarly in home preparation of food, the use of stainless steel, aluminum, and teflon have reduced the opportunities of food-lead contact to practically nothing. About the only point of contact between lead and food in industry is in solder used in the manufacture of tin cans. Schroeder and Balassa state that a small amount of lead may enter some foods from the solder on cans. The extent of leeching is dependent on the composition of the solder and the type of food. Values reported for string beans, tomato juice, and peas were 0.02, 0.06, and 0.09 ppm higher than for the corresponding uncanned product. Bergner and Miethke (8) reported that the lead uptake of the aqueous filler of canned vegetables and fruits in laquered and unlacquered tin cans during storage is very small. There is a small uptake of lead in products in cans soldered inside on the seams. Lacquering the seams reduced the lead uptake.

In summary, the available evidence indicates lead is widely distributed in all classes of natural foods, that the average daily lead intake from the diet is about 0.3 mg per day, and ranges for most people between 0.1 and 2.0. It appears also that the average lead intake from foods has not changed appreciably in the past 3 decades. (Although data are

lacking to prove the point, the consistency of these findings, coupled with the decreasing opportunity for the contamination of food by lead through agricultural application or during processing and storage, suggests the possibility that the levels of lead available in the environment may be increasing.) In view of the importance of the diet as a source of lead exposure to man and manifold factors which influence its level in food, any activity aimed at controlling the overall level of lead exposure should include continued surveillance of the diet and critical studies on the cause and effect relationships between lead in the environment and in food.

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LEAD IN DRINKING WATER

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In preparation for this symposium, data on the lead content of both raw and finished water in the United States have been gathered from various public health sources, including data generated by routine monitoring activities such as the Water Pollution Surveillance System and the monitoring of water supplied to interstate carriers. In addition, the avenue of entrance of lead into the water supply reaching the tap of the general consumer has been reviewed.

In the analysis of the data, the recently reviewed (1) "Grounds for Rejection" limit of 0.05 mg/liter for lead in drinking water has been used as a suitable guide point in considering the lead present in various waters.

Lead from Motor Boats

Lifton (2) estimated in 1963 that recreational boaters pay $50 million a year in the form of state taxes on gasoline, with the average tax being 6 cents per gallon. This leads to an estimate of about 800 million gallons of gasoline burned annually by recreational motor boats. In laboratory stuides English and co-workers (3) reported that an average of 0.53 gram of lead (about 20% of the total in the gasoline) was put into water by a gallon of gasoline burned by an outboard motor. Individual motor behavior varied in terms of the amount of lead added to the water. These two figures provide a crude estimate of about 4×10^8 grams of lead contributed by outboard exhaust at the 1963 rate of operation of the recreational outboard motor. Although this is not a significant amount of lead compared to the 8×10^9 grams estimated by Patterson (4) to be contributed by sewage, it is a factor to be reckoned with before recreational motorboating is allowed on a small waterbody that serves as a source of drinking water.

Lead Removal by Sewage Treatment Processes

Patterson's estimate (4) of the lead contribution from sewerage is recognized as being only an order-of-magnitude guess. He has not, however, credited sewage treatment with any lead removal capability. Where sewage is treated, lead is accumulated in the solids removed in the treatment processes. Studies of the composition of sewage sludges showed 0.78% of the digested sludge at Toledo to be lead and 0.215% of the excess activated sludge at Milwaukee to be lead (5). While few data are readily available on lead removal by sewage treatment processes, study of data on removal of other metals by sewage treatment processes (6) leads to an estimate of about 50% removal of lead by secondary processes and 15% removal by primary processes. Lead so removed would be spread on land or barged to sea and dumped. The fraction of the lead originating in cities that actually passes through waste disposal facilities can only be a matter of conjecture.

Lead in (Unfiltered) Surface Waters

Table 1 shows the results of analysis of a number of unfiltered water samples from the Detroit River and tributaries of that river in the Detroit area. Lead is frequently present in the river and its local tributaries in quantities well above those required to exceed the Drinking Water Standard limit. The highest value observed, 0.8 mg/liter, was found in the Ecourse River, which is reported to be essentially a local stormwater channel.

TABLE 1

Lead Content of Surface Waters in the Detroit Area[a]

(Including Solids)

Lead, mg/liter	Number samples, Detroit River	Number samples, Detroit River tributaries
0.01	53	25
0.01 - 0.019	15	8
0.02 - 0.029	29	6
0.03 - 0.039	19	2
0.04 - 0.049	11	3
0.05 - 0.059	3	1
0.060 - 0.079	5	0
0.080 - 0.100	1	2
Over 0.100		1[b]
	137	48

[a] Data furnished by D. Krawcyzk (Chief, Laboratory Section, Detroit, G L I R B). Observations made on samples collected in 1962, 1963, and 1964.

[b] 0.80 mg/liter

Lead has been determined at a number of sites by the Great Lakes - Illinois River Basin (GLIRB) project. In a recent study of the Illinois River and its tributaries in the Chicago area, 30 out of 80 samples showed more than 0.05 mg/liter of lead. The high value on the Chicago River was 0.07 mg/liter. Industrial tributaries showed values of up to 0.18 mg/liter.

On the basis of subsequent data, it appears that only a fraction of the lead in surface water is dissolved. Clearly, low turbidity is an important property which can minimize the exposure of the consumer to lead from drinking water.

Lead in Surface Waters (Dissolved)

Lead has been determined in surface waters by the Water Pollution Surveillance System since 1962. Their data have been tabulated and made available.

Before I comment on these data, it may be helpful to consider the mechanics behind the data and the analytical procedure used. These have been described in detail by Kopp and Kroner (7).

The following factors are pertinent:

1. Samples are composited over periods of 3 to 4 months and are filtered through a milipore membrane with a pore size of 0.45 micron.

2. The sample portion examined is determined by the dissolved solids content of the sample. A sample size is selected so that a processed portion of the sample will contain 100 milligrams of solids in 5 milliliters of sample.

3. The arcing procedure used yields a very flat curve in terms of scalar counts per milligram of lead until about 4 milligrams of lead is reached in the 5-milliliter portion of processed sample. The results of analysis are quite definitive for relatively large amounts of lead in the processed sample, but are very vague when the amounts of lead in the sample are small.

The combined sampling and analytical procedure yields results with the following characteristics:

1. Lead values reported are values for lead that persisted in solution or colloidal suspension for long periods.
2. Samples with the greatest dissolved content are least likely to be analyzed precisely unless the lead content is quite large.
3. For samples with low lead-to-dissolved-solids ratios, the results of the analysis are frequently not definitive and the only comment that may be made is that the lead is below a stated value.

With all but the most highly mineralized samples, reasonably well defined values emerge from the analysis unless the lead content is well below the limit stipulated by the 1963 Drinking Water Standards (0.05 mg/liter).

With this introduction, the Water Quality Surveillance System data as of October 1965 can now be considered. In all, 876 samples had been examined. Only 188 samples had sufficiently high dissolved-lead-to-dissolved-solids ratios that the analysis could arrive at a number rather than a statement such as "lead less than 0.005 milligrams per liter." Five samples showed lead in excess of 0.050 milligrams per liter. These were as follows:

Station	River	Date	Value, mg/liter
Toledo, Ohio	Maumee	11/10-18/64	0.055
Huntington, W. Va.	Ohio	12/1-31/64	0.091
New Harmony, Ind.	Wabash	10/1-12/31/62	0.057
Cincinnati, Ohio	Little Miami	4/1-6/30/64	0.053
Grafton, Illinois	Illinois	4/1-6/30/64	0.050

Before we consider the data, it is instructive to consider some of the stations whose samples did not show appreciable amounts of lead. These include points such as the Delaware and Schuylkill Rivers at Philadelphia; Hudson River below Poughkeepsie; Connecticut River at Enfield Dam, Connecticut; and the Illinois River at Peoria. At these sites the combustion of coal, the consumption of gasoline, or massive aggregations of plumbing and industry have had maximum opportunity to enrich the lead content of streams, but lead was not found in large amounts in solution.

The high values noted may be analyzed briefly. The Wabash samples at New Harmony, Indiana are all relatively high. The sample highest in lead also showed relatively large amounts of copper, zinc, and nickel, indicating the association of the high lead content with the metal-working industry. The Maumee River starts principally in the sewers of Ft. Wayne, Indiana and travels past a complex that causes it to pick up wastes from metal-working and petro-chemical operations and extensive agricultural operations. Huntington is also well situated to be immediately affected by wastes from both ferrous and nonferrous metal processing industries. The Little Miami sample is taken where both metal-working wastes and incinerator ash, along with stormwater from a large part of Cincinnati, might have affected the results.

There is no immediate explanation of the high results noted at Grafton, Illinois; however, the writer is not familiar with the situation there. In spite of the fact that large amounts of total lead start down the river from Chicago, apparently dissolved lead is relatively low at Peoria. The total lead in the river at Chicago previously discussed is enough, however, to account for the metal found at Grafton.

Lead in Interstate Carrier Drinking Water Samples 1963-1965

The Interstate Carrier Branch of the Division of Environmental Engineering and Food Protection has made available their data on the lead content of drinking water. The analyses were carried out as previously described by Kopp and Kroner (7), except that the samples were not filtered. While it is not certain that all the data available have been retrieved, the data secured are nationwide in nature and are believed to be reasonably representative of any data not included. The data are shown in Table 2.

There seems to be little basis for concern about the lead content of municipal water delivered to the citizen's tap at this time.

TABLE 2

Lead Content of Representative Drinking Water Supplies, 1963-1965

Lead, mg/liter	No. of observations
Less than 0.01	465
0.01 - 0.019	50
0.02 - 0.029	12
0.03 - 0.039	4

Lead in Rain

Several bits of data on the lead content of rain samples have been gathered. During 1964 and 1965 the Division of Air Pollution examined samples of rain collected at eight different sites. The method of collection involved using a container open only during periods of precipitation. No attempt was made to scrub solids from the container. The analysis showed 3 out of 23 samples to contain in excess of 0.050 mg/liter of lead (8).

In addition, another set of samples of rainfall was collected this year at the Robert A. Taft Sanitary Engineering Center in Cincinnati (9). Samples were collected at two different elevations; one group of samples was collected adjacent to a very busy main traffic artery, and a second group was collected on the top of a roof and about 200 feet above the highway. At the rooftop location, hood exhaust and stack gases, including laboratory incinerator stack gases, could have been a factor. The collection mechanism used included one set of operations that allowed dust to accumulate for periods of up to 11 days on the collecting surface. A second set of operations included samples less susceptible to dust accumulations. The large collecting surfaces used (18 x 28 inches) were rinsed free of dust before and after sampling, first with dilute solutions of castile soap and then with nitric acid. The lead content of the samples varied. The highest value, 0.49 mg/liter, was noted in a roof sample collected with 0.33 inch of rain over a period of 3 days. The corresponding sample at the lower sampling site showed 0.21 mg/liter of lead. Five out of 12 observations showed lead in excess of 0.10 ppm. The largest values clearly were associated with maximum opportunity for inclusion of dust.

The total impact of these data on lead in rain is quite simple. The cistern user who lives in a populated area or close to a major highway seems to be the individual most menaced by lead in the water he starts out with.

Lead in Direct Contact With Drinking Water

Lead or lead compounds come into direct contact with drinking water in a variety of ways. Lead chromate paints are favored by the water works industry for painting the inside of steel water-storage tanks (10, 11).

The most recent "Report of the Public Health Service Technical Committee on Plumbing Standards" (1962) lists lead as a material suitable for "water service pipe" (i.e., conveying water to a building). Interestingly enough, the same report states that "piping conveying water shall be constructed of non-toxic material."

Copper tubing is one of the more popular means of conveying water in water distribution service systems (i.e., water within a building). This is almost invariably soldered in a multiplicity of places with lead-solders.

There is an abundance of evidence that lead derived from sources within the water distribution system has caused both morbidity and mortality (12). There are no clear and unequivocal rules for delineating the boundaries of conditions under which measurable amounts of lead cannot be picked up from water handling systems.

Questions for Consideration by the Conference

Appropriate questions for consideration by this conference appear to be: Should the existing drinking water standard on lead be reviewed as to the lead content permissible in potable water?

Has the availability of alternative, economical, and nontoxic materials progressed to the point where lead paints and lead pipe may prudently be excluded from systems handling drinking water?

Should the conference indorse:

1. Studies of the pickup of lead from soldered copper tubing installations conveying water of various compositions under various conditions?
2. Studies of exposure of the citizen to lead from water in older housing that still has major amounts of lead piping?
3. Field studies of the exposure of the cistern user to lead from the water supply?
4. Intensified surveillance of interstate carrier water supplies?

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RECENT HISTORY OF LEAD EXPOSURE IN U.S. INDUSTRY, 1935-1965

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INTRODUCTION

The term "recent history" is used to include the 30 years prior to 1965 for three reasons. 1. The dithizone method for lead analysis came into general use around 1935. 2. The use of the dithizone method provided more accurate and thus more comparable results from laboratory to laboratory. 3. The period 1935 to 1965 brackets the time before and after the hygienic standards for lead came into use (1943-1945), that had a profound influence on controlling industrial lead exposures.

Except for 2 Studies, all of the data to be reported here is presented for the first time. The data are concerned exclusively with lead concentration in air and in urine of workers. ATMOSPHERIC load levels to be reported date from 1935 to 1940 and comprise,

1. An automobile manufacturing plant. (Hyler & Bradley, 1937). (Table 2).
2. Eight storage battery plants. (McCord et al. 1937). (Table 3).
3. Twelve nonferrous foundries (Detroit Dept. of Industrial Hygiene, 1940). (Table 4).

URINARY lead levels to be presented concern 7 work categories that represent the major uses of land.

1. Automobile, manufacture and repair (Table 5).
2. Storage battery, manufacture and rebuild.
3. Foundries.
4. Electrotype printing.
5. Secondary smelters.

The data speak for themselves. The data show in a clear, consistent and unmistakable way that in every work category examined there has been a steadily decreasing exposure to lead in industry since the hygienic standards were introduced in 1943, following a tremendous decrease beginning after 1937.

Slide 1. Consumption of Lead in U.S. Slide 1 shows the consumption of lead in the U.S. in millions of pounds for each decade to date since 1934 according to major uses that are soon to be discussed. Slide 1 shows that the total consumption of lead has increased about 2 1/2-fold since 1934. This 2.5-fold increase was about that for use in STORAGE BATTERIES, TYPE METAL and for gasoline additives since 1944. The largest increase of 10-fold was for use in automobile body manufacture. With the exception of ammunition these are the categories on which human exposure data are to be presented.

Slide 2. Air analysis for Lead in Automobile Body Manufacture, '37. Slide 2 shows that of seven major work categories in automobile body manufacture in 1937 there was no single operation that did not exceed the hygienic standard of lead for air. Indeed it was this study, among others, that precipitated the development of the air standard for lead.

Slide 3. Air Analysis for Lead in Storage Battery Plants, '37. Slide 3 shows an almost similar condition existing in the air that workers breathe in STORAGE BATTERY plants, at about the same time (1936-1937); only one plant had levels that did not exceed the subsequently set TLV by factors of from 2 to 40.

Slide 4. Lead Levels in Foundry Air. Slide 4 shows that the lead levels in the FOUNDRY-ROOM air at about the same period (1940) exceed the threshold limit value subsequently to be set, in 9 plants of 12 in which air samples were taken. Excesses over the TLV, of 2- to 5-fold, were much less than that in the automobile and battery building plants.

Slide 5. Comparison of Urinary Lead Values in Five Work Categories in 1965, vs. 1945. Slide 5 shows the percent frequency distribution of 1820 urinary lead values. In every work category it is clearly evident that there has been not only an appreciable reduction in mean urinary lead values during the past two decades, but that the total number of specimens in excess of the biologic threshold limit has appreciably decreased. And as a consequence the numbers exceeding the biologic threshold limit have materially decreased.

Slide 6. Comparison of Urinary Lead Values in Storage Battery Plants. Slide 6 is presented in all fairness to show that not all plants conform to the general trend of decreased lead exposure with the years. On the left side of the slide, however, are the analyses showing that all battery plants surveyed in the Detroit area conform to the general trend. The plant in the right column represents a special instance. When this plant started up in 1952 it already had initially very good control of lead exposures. Subsequently, owing to a building unification process, exposures from the high-exposure areas have become mixed with those presenting low exposures, making the over-all exposure to lead somewhat higher than in the past. The mean urinary lead levels are still within acceptable limits.

CONCLUSIONS

A review has been made of mostly hitherto unassembled and unpublished data on lead exposure as measured by concentrations in air and in urine, in 7 of 8 work categories representing the major uses of lead: auto body, collision and manufacture; foundries, printing (electro-type); storage battery manufacture and rebuilding; secondary smelters. The large category of munitions is excepted.

Insofar as the concentration of lead in air and in urine can be used as measures of exposure to inorganic forms of lead, the clear, consistent, and unmistakable conclusion is that exposure to air-borne lead, dust and fume, is less by a factor of several magnitudes in all industrial categories since 1934. This decrease has continued, but at a slower rate since 1945, at which time the average exposure in most work categories was below the acceptable limits owing to the introduction of safe work standards for lead in air and in urine.

Of a ninth work-exposure category, involving exposure to gasoline additives and their breakdown products (organic and inorganic lead) which includes gasoline service station attendants, refinery handlers, garage mechanics, parking attendants, traffic officers and motor vehicle drivers, only one, GARAGE MECHANICS, showed evidence of exposure (elevated urinary lead values) slightly in excess of normal (1955, Kehoe), but still well below acceptable limits.

TABLE 1

CONSUMPTION OF LEAD IN U.S. *
(millions of pounds)

	1964	1954	1944	1934	Fold Increase
Gasoline	448	320	170	-	2.6 since '44
Storage Battery	859	674	614	326	2.5
Automobiles (Solder)	142	142	41	14	10
Ammunition	113	80	126	70	
Type Metal	51	52	46	26	2.0
Brass & Bronze	46	40	-	-	
Other: bearing, pigment, cable cover, etc.	745	882	123	540	
Total	2,404	2,190	1,120	976	

*From Bureau of Mines Minerals Yearbooks.

TABLE 2

AIR ANALYSIS FOR LEAD IN AUTOMOBILE BODY MANUFACTURE (1937)* in mg/m^3

TLV 0.15 mg/m^3 (1946)

Process	No. Samples	Concentration Mean	Concentration Range	Fold increase in mean over TLV
Soldering	9	3.4	0.3-7.0	23
Soldering & Tinning	3	5.5	5.0-6.0	37
Metal Finishing	7	5.5	0.6-15.2	37
Soldering & Finishing	5	4.9	1.6-10.3	33
Assembling	9	2.2	0.5-8.5	15
Door-Hanging	5	6.3	2.6-12.3	49
Inspecting	12	1.0	0.4-1.9	7
Miscellaneous	11	2.8	0.2-5.4	18

*From Hyler & Bradley, Ind. Med. April 1938.

TABLE 3

AIR ANALYSES FOR LEAD IN 8 STORAGE BATTERY PLANTS (1936-1937)*

Plant No.	mg Pb/m^3 Mean	mg Pb/m^3 Range	% samples Above TLV	Plant No.	mgPb/m^3 Mean	mgPb/m^3 Range	% samples Above TLV
1	5.8	0.4-25	100	5	0.74	0.05-2.3	75
2	0.9	.7-1.2	100	6	0.9	.56-1.7	100
3	0.3	.18-.49	75	7	0.3	0.2-0.4	75
4	0.18	.06-.2	0	8	0.34	0.1-0.7	72

*From McCord, et al., Ind. Med. 6, 362 (1937).

TABLE 4

LEAD LEVELS IN FOUNDRY ROOM AIR IN mg/m^3 (1940)*

Plant	Furnace RM		Foundry Gen'l air		Foundry pourings		Miscellaneous grinding, core RM et al.		Mean exposure	In excess of limit
	Mean	Range	Mean	Range	Mean	Range	Mean	Range		
1	0.23	.08-.39	-	-	0.23	.16-.35	.075	.07-.08	0.18	No
2	0.43	.19-.71	.36	.29-.4	-	-	.11	.04-.19	0.30	Yes
3	-	-	.95	.95	-	-	.69	.55-.80	0.82	Yes
4	0.52	.13-.86	.96	.83-1.1	1.2	-	.28	-	0.74	Yes
5	0.71	.31-1.45	.95	.5-1.4	1.2	.73-2.1	.83	.04-.13	0.82	Yes
6	0.55	.16-1.1	.3	-	.43	.16-.71	-	-	0.43	Yes
7	0.33	.2-.45	.16	-	1.2	-	.36	.04-.74	0.51	Yes
8	0.79	.005-.23	0.04	.03-.05	.02	.005-.04	-	-	0.28	Yes
9	0.13	-	-	-	.22	.21-.22	-	-	0.18	No
10	0.09	-	0.09	-	.08	.06-.1	-	-	0.09	No
11	0.82	.79-.85	-	-	0.5	.3-.9	-	-	0.70	Yes
12	0.72	.4-1.0	.5	-	0.65	.45-.94	-	-	0.52	Yes

* Unpublished data through the courtesy of Dr. Wm. G. Fredrick, Dir. Detroit Dept. of Industrial Hygiene.

TABLE 5

PERCENT FREQUENCY DISTRIBUTION OF PERSONS IN VARIOUS OCCUPATIONAL GROUPS
According to Urinary Lead Values for the Periods 1945-1950 and 1960-1965*

Lead in urine mg/l	Auto body				Foundry		Printing		Sec Smelter		
	Collision		Mfr.				Electrotype				
	'45-'50	'60-'65	'45-'50	'60-'65a)	'45-'50b)	'60-'65c)	'45-'50	'60-'65	'45-'50	'60-'65	
0.00-.10	55	75	51	69	25	32	68	86	27	44	
0.11-.20	27	21	34	18	37	50	25	10	32	35	Biol.
0.21-.30	17	3	12	8	21	14	3.5	3.5	18	11	TL
0.31-.40			1.4	2	9	3	3.5		8	4	
0.41-.50	1	0.5		1	6	0.7			8	3	
0.51-.60		0.5	1.4	1	1	0.7			2.5	1	
.61					1	0.2			3.9		
Total Nos.	64	193	73	165	245	558	57	140	158	167	1820
MEAN	0.11	0.08	0.13	0.10	0.19	0.15	0.10	0.06	0.22	0.15	
RANGE	0.1-.44	.01-.51	.03-.53	.01-.52	.01-.68	.01-.64	.02-.35	.01-.26	.01-.61	.01-.56	

*Data through the courtesy of Dr. Wm. G. Fredrick, Detroit Dept. of Ind. Hygiene.
a) 1955-1963
b) 1948-1949
c) 1963-1965

TABLE 6

PERCENT FREQUENCY DISTRIBUTION OF STORAGE BATTERY OPERATIONS ACCORDING TO Urinary Lead Values Past (1945-1954) and Present (1960-1965)

Lead in urine mg/1	Several small plants				One large plant (600)		
	Mfr.		Rebuild		Mfr.		
	'45-'50	'60-'65	'45-'54	'60-'63	'53-'54	'63-'65	
0.00-0.1	21	28	31	54	80	53	
0.11-0.2	28	36	34	39	18	35	Biol.
0.21-0.3	24	22	16	7	1.3	8	TL
0.31-0.4	13	14	8		.2	2	
0.41-0.5	4		9.6			.6	
0.51-0.6	3					.2	
.61	7		1.2			.2	
TOTAL NOS.	194	170	83	76	526*	1,142*	2,191
MEAN	0.24	0.19	0.20	0.10	0.07	0.12	
RANGE	.01-.97	.02-.82	.02-.98	.01-.27	0.01-0.24	0.01-0.56	

* Approx. 10% sample of available data.

COMMENTS AND DISCUSSION FROM THE FLOOR

After the conclusion of this paper, Dr. Kehoe rose to point out that its conclusions may be spurious because much of the data on which they were based originated from Detroit which has had unusually good industrial hygiene control and thus may be untypical of the rest of the nation. Dr. Hardy rose to make a similar statement indicating essential agreement with the remarks of Dr. Kehoe, pointing out that in many small shops overexposure to lead was still common.

In my (Dr. Stokinger) rebuttal to both these remarks I pointed out that while it is recognized that today there is still overexposure to lead in small industries and shops, it is a question of relative numbers and I seriously question whether the summated numbers of those overexposed in small shops were anything more than a relatively small segment of the total exposed lead workers, thus maintaining the essential verity of the conclusions. Numbers of lead workers broken down in this way were not available at the meeting.

* * * * *

PANEL: RISK OF EXPOSURE AND ABSORPTION OF LEAD

Chairman: J. H. Sterner
Co-Chairman: H. Heimann

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UNDER WHAT CIRCUMSTANCES IS INHALATION OF LEAD DANGEROUS?

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LUNGS AS A PORTAL OF ENTRY

The lungs can serve as a portal of entry for numerous substances. In order to understand the entrance, deposition, and effect of inhaled particles such as lead it is necessary to understand the anatomic physiology of the respiratory system and to know the nature and concentration of the material inhaled.

ARCHITECTURE AND PHYSIOLOGY OF THE RESPIRATORY SYSTEM

Figure 1 is a sketch showing areas of the lungs of particular interest in particle deposition and retention. Figure 2 is a photograph of a plastic cast of the bronchial and vascular tree of adult human lungs. In essence the lungs are a vast capillary network separated from the alveolar spaces by a delicate submicroscopic membrane. Both air and blood are delivered to the alveolar capillary bed through a complex branching tree arrangement. The conducting portion of the lungs, the airways, is made up of 23 or more generations of dichotomous branching before it terminates in the alveolar spaces. These alveolar sacs are not seen on the cast but are shown in Figure 1. The alveoli are approximately 400 microns in diameter and number over 300 million. There are also 14 million alveolar ducts and 280 billion capillary segments. The alveolar capillary surface is therefore between 40 to 80 square meters, depending upon the size of the lungs. Soluble material depositing on the alveolar surface can therefore be readily absorbed and approximate an intravenous injection without local reaction. The alveolar membrane is very thin. Substances readily pass through the alveolar lining, the interstitial tissue, and the capillary wall into the circulating blood. Absorption of material occurs at the alveolar capillary level. Little if any absorption occurs into the general systemic circulation from deposition on the trachea or bronchi. There is perhaps some local absorption as demonstrated with therapeutic aerosols and local anesthetics. (1)

To sustain life, the average adult must breathe about 450 cubic feet of air a day. This volume of air contains approximately 1000 billion particles, with a total weight of 1-2 milligrams. Because the lungs behave like a viscous filter, a large percentage of these particles are deposited along the most tortuous passages. Some may get down to the alveoli. The effectiveness of this filtration system can be appreciated by the fact that secretions from the normal tracheobronchial tree are sterile.

MECHANISM OF DEPOSITION AND RETENTION OF INHALED PARTICLES

The amounts of aerosol particles, such as lead, deposited in the lungs and the site of their deposition is dependent upon the following factors: (1) particle size and density; (2) the hygroscopic nature of the particle; (3) certain physiologic functions, such as the rate and depth of breathing; and (4) duration of exposure. Generally, substances of high solubility are rapidly and completely absorbed by the respiratory epithelium. Highly soluble and irritating substances, even of small particle size--under 5 microns, will produce an immediate reaction in the upper respiratory passages producing cough, dyspnea, and hypersecretion. Intermediately soluble and less irritating substances of the same size will deposit and react in the lower respiratory system, bronchi, bronchioles, and alveoli. The nature of the material and whether it is soluble in water or lipids will determine the absorption rate in the respiratory system. Temperature also has a bearing, in that cooler particles because of thermal deposition effects will descend further into the respiratory tree.

Insoluble particles depositing along the tracheobronchial tree become imbedded in the "mucus blanket" covering the ciliated epithelium. The cilia propel the mucus and particles against gravity up the tracheobronchial tree to the pharynx from whence it is either swallowed or expectorated. While the "ciliated escalator" is the major cleansing mechanism of the tracheobronchial tree, other factors such as cough, secretion of mucus, and peristaltic action are important also.

Particles reaching beyond the terminal bronchioles where cilia do not exist may deposit, persist, undergo dissolution, be absorbed, or be engulfed by alveolar phagocytes. From here they can enter directly into the interstitial spaces, lymphatic channels, or leave the alveoli via the alveolar ducts, enter into the bronchioles to be propelled along the ciliated mucus stream. Whether particles have a damaging effect on the lungs depends upon their physical and chemical nature, the sensitivity of the individual, and any irritating effects of the particles--more about these aspects later, in reference to lead.

The mechanism of particle deposition in the lungs has been a subject of considerable theoretical investigation. (2) Experimental studies have been made in reference to cigarette smoke; however, not much empirical information is available. From the theoretical and experimental studies, it can be concluded that particles are deposited in the respiratory system by one of the following three mechanisms:

1. Inertial Impaction: A particle suspended in an air stream moving through the passages of the respiratory tract is not always able to follow the air stream throughout because of the inertia of the particles. Consequently, the particles deposit against the walls of the passages. This is especially true in the upper respiratory passages (the nasal airway, pharynx, and larynx) where air velocity is high. The inertial effect on particles is directly proportional to their density and the square of their diameter.
2. Sedimentation: Because of gravitational effects, aerosol particles, except for extremely small ones, settle downward. This mechanism is one of the more important affecting larger particles entering deeper regions of the lung where air velocity is reduced. The sedimentation rate is proportional to the particle density and the square of the diameter. Therefore, the more massive particles which have not deposited by inertial impaction will be deposited by sedimentation. The settling rate for relatively small particles can be calculated from Stokes Law.
3. Brownian Motion: In the alveoli where air velocity is quite low, the alveolar walls being the dead end of the respiratory tract, Brownian movement becomes an important mechanism of particle deposition. Very small particles which become suspended in the air are in continuous motion because they are constantly being bombarded by air molecules. The mean displacement after a given time can be computed from Einstein's diffusion equation. From this equation, rate of removal of particles by diffusion can be calculated.

Theoretical studies of particle deposition have been made and calculations reported by Findeisen (3) and Landahl (4). A plot showing calculated and measured retention of particles obtained by Landahl (5) and Altshuler (6) is shown in Figure 3. This Figure indicates that the retention is at a minimum for particles approximately 0.3 micron in diameter and increases for smaller and larger particles. A comparison of data obtained from three different investigators is presented in Figure 4.

From the particle deposition data available, it can be safely stated that particles larger than 50 microns fail to traverse the respiratory tract beyond the major bronchi. Particles 5 microns or smaller descend to the level of the respiratory bronchioles and alveolar ducts. Particles 0.6 micron or less reach the alveoli. According to the data of Wilson and LaMer, maximum alveolar retention occurs with particles of 2.0 microns down to 0.3 micron--see Figure 4, a comparison of lung retention data of Altshuler, Landahl, Wilson, and LaMer (2). Retention of particles by the lungs, in addition to particle size, is dependent upon respiratory frequency. The slower the respiration, the greater the residence time, and, consequently, the higher the retention.

A study has been made of the deposition of aerosols of cigarette smoke in the respiratory system. (7) Smoke is an extremely concentrated aerosol of tar droplets with a mass median diameter of about one micron; there are approximately 100 billion such droplets in an average 35-cc puff. Depending upon smoking habits, somewhere between 25 and 98 percent of the mass of these particles is deposited in the respiratory tree. Figure 5 shows the relationship of the recovery of cigarette smoke from the respiratory tract to mean particle size.

Studies employing a local anesthetic with a mean particle size of 5 microns have resulted in satisfactory short-term oropharyngeal and tracheobronchial anesthesia for bronchography and oropharyngeal intubations without systemic effects indicating selective deposition based mainly on particle size. (1)

Factors which interfere with the cleansing mechanisms of the lungs are: (1) air pollutants (for example, cigarette smoke is known to paralyze ciliary activity and interfere with drainage); and (2) diseases, such as bronchitis and emphysema. Emphysema with its parenchymal destruction causes increased particle deposition; emphysematous areas of the lungs are known to contain more pigment and foreign material than in the better ventilated and normal areas. The more severe the emphysema, the more severe the effects of air pollutants. Air pollutants are said to irritate and aggravate the symptom complex of emphysema and bronchitis.

INHALATION OF LEAD

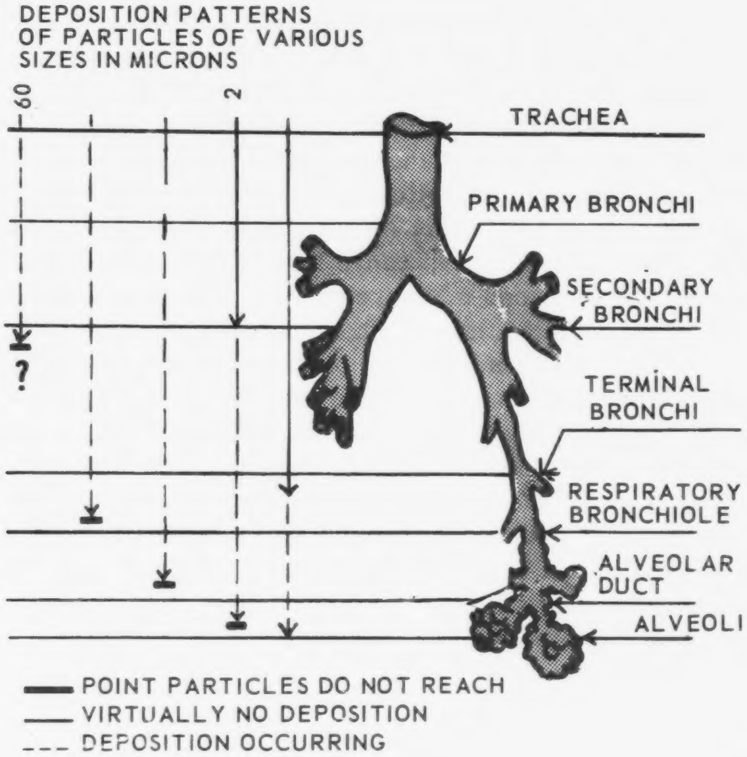
Sources of air contamination of lead can be listed as follows:

1. pesticides.
2. distillation or burning of coal.
3. incineration of refuse.
4. industrial.
5. natural.
6. dusts and sprays.
7. tobacco smoke.
8. paint attrition.
9. motor vehicle exhausts.

Lead occurring naturally in soils and rocks contributes only a small part to air pollution. In industrial areas and where distillation and burning of coal occurs, higher atmospheric levels can be attained. Lead can be emitted into the atmosphere from smelting processes, from the incineration of scrap and other industrial uses, and from dusts and sprays (insecticides containing lead). The most important source of lead contamination of the atmosphere is exhaust from vehicles using leaded gasoline to slow down combustion. The inhaled organic form of lead is most harmful since it is soluble in lipids and other body fluids. Inorganic lead is relatively insoluble and for the most part should be poorly absorbed from the respiratory tract. The cleansing mechanisms described previously remove the majority of the inorganic lead particles inhaled. These particles are then either expectorated or swallowed. Atmospheric levels of lead have shown that the concentration of lead in the Los Angeles area is around 5 micrograms per cubic meter. In other areas it is stated to range between 1.7 to 2.5 micrograms per cubic meter. (8) The particle size of lead measured in the Los Angeles area is said to be of such magnitude that it reaches the alveoli where deposition, retention, and absorption via the lungs can take place. Studies of Robinson et al (9) show that 60 percent of the atmospheric lead in the Los Angeles area is less than .45 micron, 75 percent less than .90 micron, and 90 percent less than 1.60 microns. Particles larger than 2 microns would impinge on the airways to be cleansed and swallowed and for the most part are handled as lead ingested by mouth. Studies on cigarette smokers indicate higher blood levels of lead than in nonsmokers, in part due to lead contained in the tobacco, the interference with cleansing mechanisms, and high particle deposition and retention of cigarette smoke. There is no known immediate or local effect of inhaled lead on the respiratory mucosa and no evidence to suggest the lead is a pulmonary carcinogen.

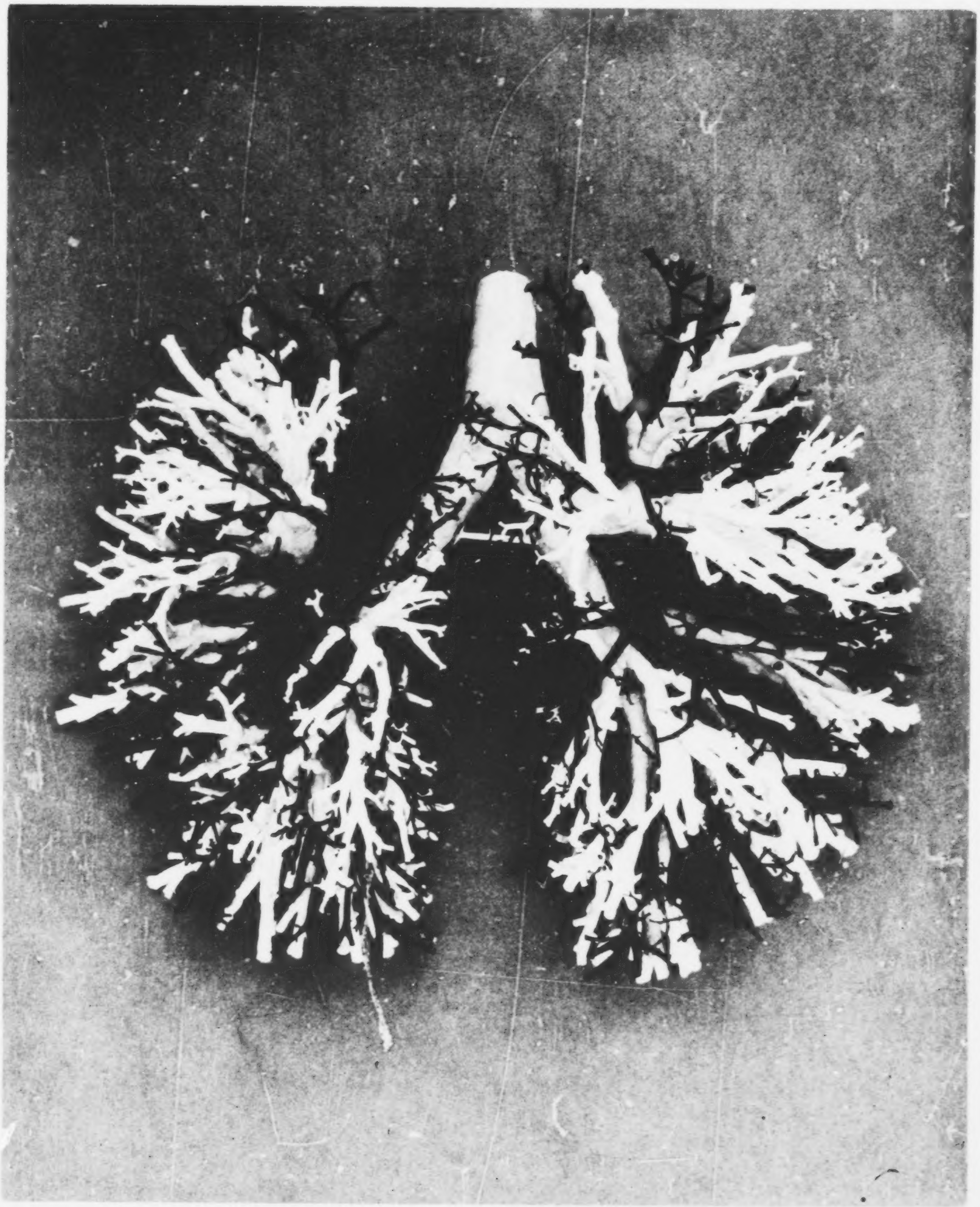
SUMMARY AND RECOMMENDATION

It appears that the majority of inhaled lead is removed or cleansed from the tracheobronchial tree by the natural cleansing mechanisms and swallowed or expectorated. Whether lead is taken up by the lungs depends upon solubility factors and particle mechanics. Highly soluble lead in proper particle size such as fumes or mists can rapidly be absorbed and approximate an intravenous injection. A possible unproven relationship exists between pulmonary disease--bronchitis and emphysema--with particle deposition, cleansing, and accumulation of lead. There is no known direct effect of lead on the bronchopulmonary system. There is need for further determination of the concentration and particle size of lead in the atmosphere. Studies on particle retention and deposition of lead in the lungs are lacking. The synergistic effect of lead with other pollutants such as carbon monoxide and sulfur dioxide is not known. This is especially true in the patient with chronic obstructive pulmonary disease. It would seem that patients would be more susceptible to lead inhaled because of greater retention and increased deposition. Studies are needed to determine the concentrates of lead in the tissues and fluids of the lungs. Whether lead is immobilized from body stores in diseased states such as respiratory acidosis remains to be proven.


DEPOSITION PATTERNS
OF PARTICLES OF VARIOUS
SIZES IN MICRONS
60
2
?
TRACHEA
PRIMARY BRONCHI
SECONDARY BRONCHI
TERMINAL BRONCHI
RESPIRATORY BRONCHIOLE
ALVEOLAR DUCT
ALVEOLI
POINT PARTICLES DO NOT REACH
VIRTUALLY NO DEPOSITION
DEPOSITION OCCURRING

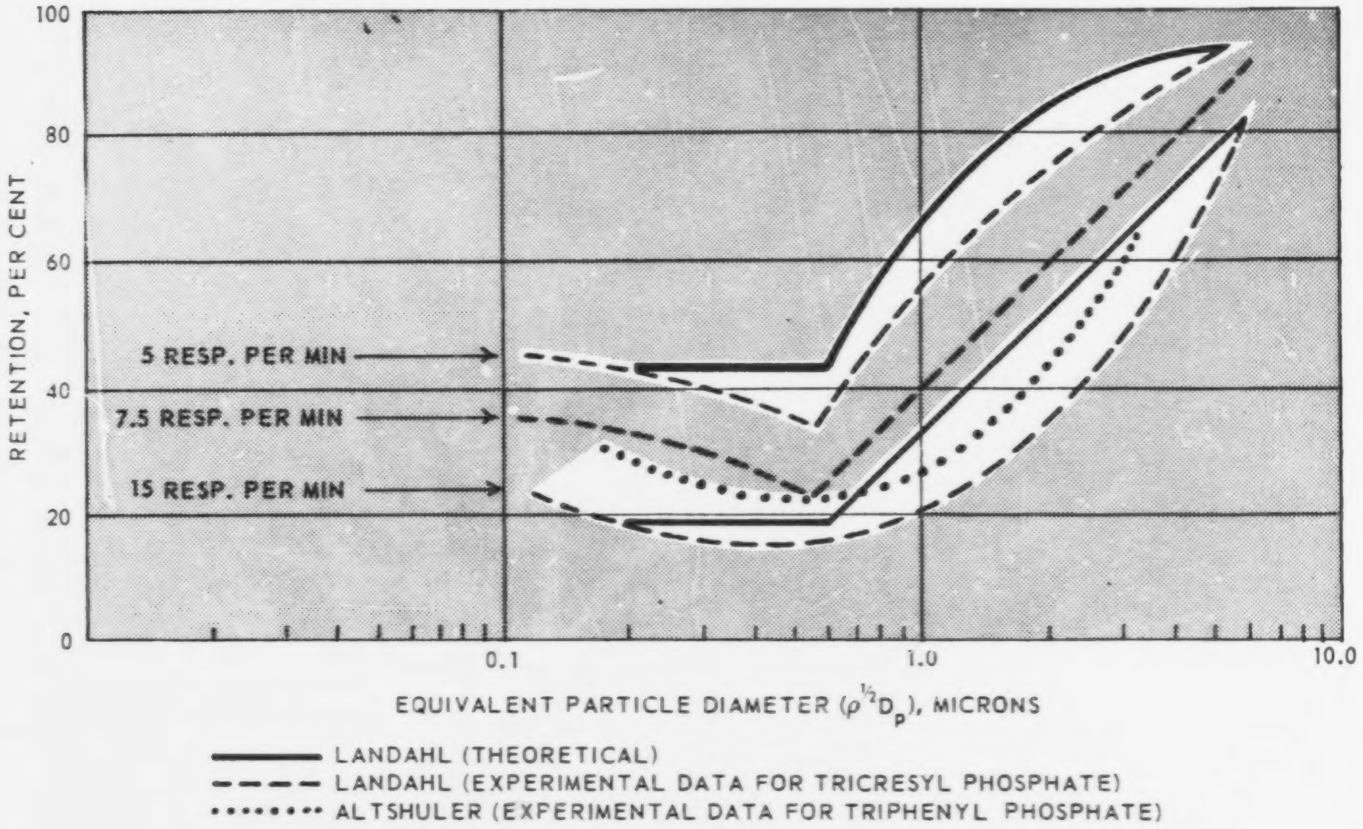
A SCHEMATIC REPRESENTATION OF THE AIRWAYS AND SITE OF DEPOSITION OF PARTICLES IN ACCORDANCE TO SIZE AFTER VALUES COMPUTED BY LANDAHL

FIGURE 1.



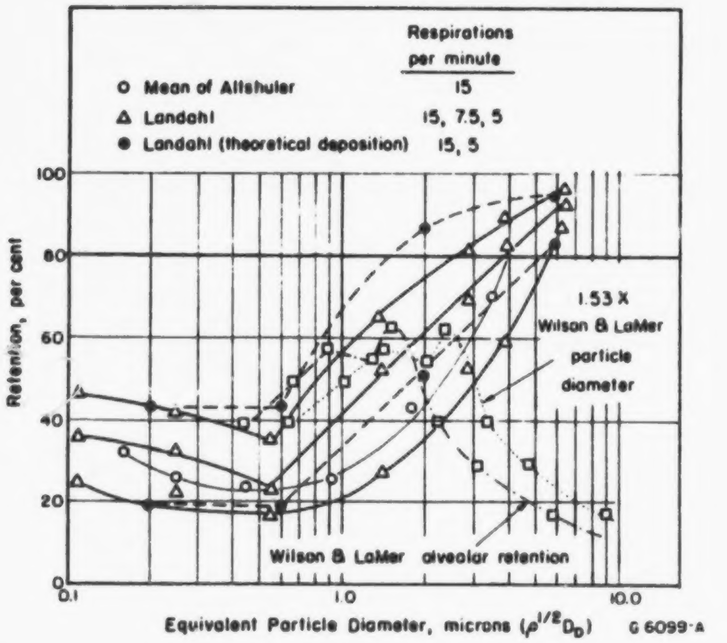
PHOTOGRAPH OF A PLASTIC CAST OF THE BRONCHIAL AND VASCULAR TREE OF AN ADULT HUMAN LUNG

FIGURE 2.



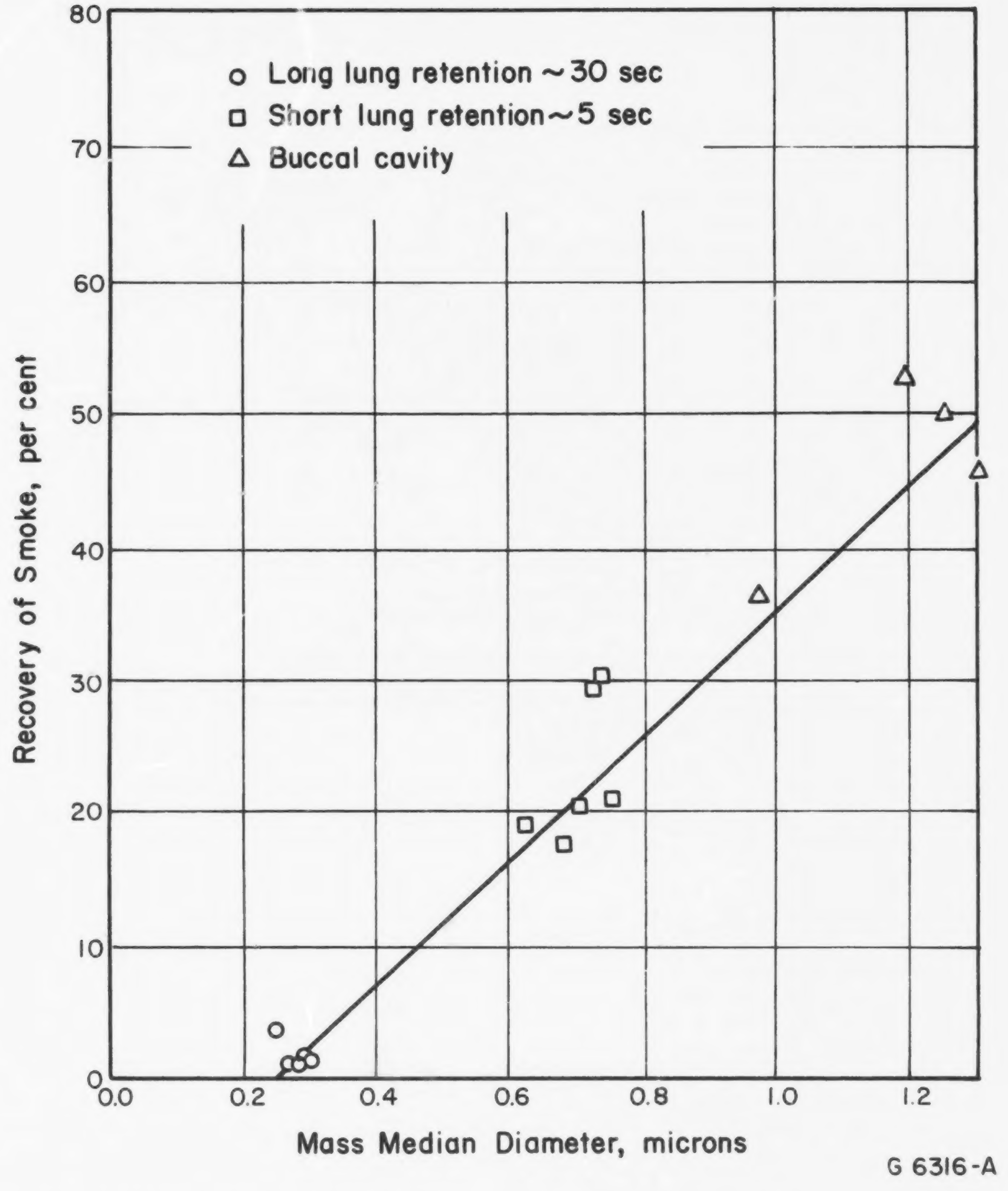
A PLOT SHOWING THE CALCULATED AND MEASURED RETENTION OF AEROSOL PARTICLES OBTAINED BY LANDAHL AND ALTSHULER. THESE FIGURES SHOW THAT RETENTION IS AT A MINIMUM FOR PARTICLES 0.3 MICRON IN DIAMETER AND INCREASES FOR SMALLER AND LARGER PARTICLES. TOTAL LUNG RETENTION SHOULD NOT BE CONFUSED WITH ALVEOLAR DEPOSITION OR ASSUMED TO MEAN TOTAL ABSORPTION.

FIGURE 3.



Comparison of lung retention data obtained by three different investigators

FIGURE 4.



RELATION OF RECOVERY OF CIGARETTE SMOKE FROM THE RESPIRATORY TRACT TO MEAN PARTICLE SIZE

FIGURE 5.

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UNDER WHAT CIRCUMSTANCES IS INGESTION OF LEAD DANGEROUS?

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The discussions of this meeting are concerned with the degree of risk of lead poisoning, if any, to which persons in the general population are subjected through the occurrence of lead in their general environment. In examining this problem, it is first necessary to differentiate between general environmental conditions and those which have long been known to be potentially hazardous in certain lead trades and lead-using industries. It is also necessary to recognize certain other situations in which lead poisoning occurs, when, through innocence, ignorance, or irresponsibility, the non-occupational environment of an individual, a family, or some other group may be rendered dangerous by personal behavior, or through the utilization of materials or equipment commonly known to be dangerous. An example of unusual behavior is that of the infant, usually poorly cared for, who habitually, for weeks or months, eats dangerously large quantities of lead-containing paint from the surfaces of outer or inner walls, woodwork, furniture, or toys, in his home. Further examples are furnished by the owner of an isolated cabin or camp, or even a permanent home, who conveys water into his house from a spring or other source through a long stretch of lead pipe; or by the frugal collector of maple sap who preserves his wooden pails against disintegration by coating them generously, inside and out, with a paint high in its lead content; or by the art-conscious but technically incompetent potter who puts out a badly glazed brand of tableware with which to beguile aesthetic housewives into feeding their families dangerously.

The foregoing are but a few examples of serious dangers associated with the ingestion of lead compounds, as variants of a host of recorded and assembled (1, 2, 3) human experiences which, collectively, have been responsible for numberous and sometimes fatal cases of non-occupational lead intoxication-incidents and epidemics which have come and gone over the centuries, some of which have been investigated and elucidated in times past and present. But while some of these have been informative with respect to the quantitative relationships between dosage and time in the induction of lead poisoning, it is only in these respects that they are pertinent to the subject under discussion here. For the object of present concern is not the incident or the mischance that has yielded the unusual case of lead poisoning within the general population, but is, rather, the prevalent, regular exposure to and absorption of lead, which is the common lot of people who live in the environment of a modern technological nation.

The question, as I choose to restate it for my purposes, is threefold. What are the facts with respect to the magnitude of the total exposure to lead to which citizens are being subjected? Is such exposure hazardous to a certain segment (or segments) of the population now or in the near future? If not, what margin of safety exists, or should exist? I am scheduled to speak only of the hazard of ingested lead, and I shall fulfill this mandate. However, it must be evident to all of us that this is but one facet of the matter at issue, and that it cannot be dealt with at this or any other time independent of the respiratory absorption of lead. Therefore, in order to determine the threshold value of the alimentary factor in relation to public safety, the respiratory component must be held constant, and vice versa. This, for all practical purposes, is what has been done in an experimental approach to this matter.

THE TOTAL MAGNITUDE OF THE CURRENT EXPOSURE OF THE GENERAL POPULATION TO LEAD

In the course of a series of investigations carried out in the Kettering Laboratory during the past forty years, but more specifically, those of the past twenty-eight years (because of the essential uniformity of the analytical method employed during that period), it has been possible to visualize in a fairly comprehensive manner the daily contribution made to the alimentary intake and absorption of lead by the food and beverages consumed generally (4, 5). By indirect measurements on the one hand (5), and by calculations based on physiological parameters on the other, a reasonably satisfactory approximation of the quantities of lead breathed from the ambient atmosphere by adults in the population has also been arrived at. (The latter quantities vary from time to time and place to place, and to some extent from individual to individual, as do, also, the quantities ingested in food and drink. There is no present means, however, by which the respiratory intake can be measured directly so as to obtain a statistically valid array of findings. Therefore, they must be expressed as a range of estimated values for which, at present, there is little virtue in an average value. The range, however, is not extensive in the absolute sense, and there is little doubt that the order of magnitude encompassed thereby is realistic.)

The food and beverages consumed daily by an adult in the U.S. contain lead in amounts which range from somewhat less than 0.1 to 4.0 milligrams (occasionally more). The lowest and highest values occur infrequently, the large proportion of the quantities grouping themselves regularly around the mean value of approximately 0.3 mg. per day (this mean value is derived from a large number of results obtained from a sizeable group of adults); the mean values for individuals obtained by daily observations over periods of months or years, range from 0.12 to about 0.38 mg. per day, their variance being due to the differences in the gross quantities and the choices of food and beverages consumed, habitually, by different individuals of different size, occupation, and appetite.

The quantities taken in daily with the inspired air may range from somewhat less than 0.015 to 0.09 (15 to 90 micrograms) per day, (no doubt, being considerably closer, as the usual experience, to the lower than to the higher value), depending upon where and in what manner one resides and where and how he works; i.e., upon the quality of the air breathed during the 24 hours of each day, with respect to lead content, and upon the rate and depth of the respiration during sleep, work, and recreation.

The quantities of lead absorbed into the human body from the sources indicated above may be estimated with fair accuracy on experimental evidence which shows that somewhat less than 10 percent of that ingested under ordinary circumstances is absorbed daily from the alimentary tract, while from 25 to 50 percent of that inhaled (5), dependent upon the size, shape, and density of the particles inhaled, is retained and absorbed in the respiratory system. The average quantity absorbed daily in the alimentary tract of the adult is of the order of 0.03 milligram (30 micrograms), while a reasonably satisfactory estimate of that absorbed in the respiratory system would not be far from 0.2 milligram or 20 micrograms (5, 6).

THE SIGNIFICANCE OF THE CURRENT EXPOSURE OF THE GENERAL POPULATION TO LEAD, FROM THE ASPECT OF HAZARD

If there is to be a satisfactory answer to the question whether the current trend in the absorption of lead by the general population is hazardous, the answer must come, it seems, from quantitative, physiological information as to the fate of lead absorbed over long periods of time (extended, in the final sense, to the human lifetime), and from the facts of toxicology and clinical medicine, as to the conditions under which intoxication by lead occurs.

First, with respect to the metabolism of lead taken in and absorbed by typical representatives of the healthy, adult population, the facts, while seemingly not so comprehensive, in relation to a population of nearly two hundred million people distributed throughout the U.S., as to cover all of the possible environmental variations, are so uniform, physiologically, as to be convincing. The results of experiments carried out under exacting conditions in the laboratory, supported in breadth by extensive sampling of selected and random groups in the population, have demonstrated: (a) that the intake of lead by typical, healthy, male adults is balanced for all practical purposes by an equivalent output; (b) that there is no progressive increase over prolonged periods of time in the rate of the excretion of lead from the body, nor in the concentration of lead in the blood; and (c) that any increase in the concentration of lead in the tissues of the body, including the skeleton, which may occur over the span of life is too small to be measured, being within the limits of the individual variability in the intake and output of lead (4, 5). (The latter statement should not be misinterpreted to mean that the total quantity of lead found in the body does not increase with growth from childhood to adult life). The increase observed, however, is not accumulation, in the physiological sense, but is, rather, the maintenance of a balanced concentration of lead in individual tissues, that is to say, the maintenance of an equilibrium with the environment.

Second, with respect to whether the concentration of lead in the tissues of people, generally, and the quantities in their entire bodies and in the metabolic processes described above, constitute a risk--the risk of intoxication by lead in any form known to physicians, physiologists and pathologists--the answer is in the negative. The toxic effects of lead upon the human body, as expressed in the more severe types of plumbism known classically both before and since the days of Tanquerel des Planches (7), have not been seen frequently in recent years, but they still occur from time to time, especially among children who display the immediate signs and symptoms and certain sequelae of the most damaging forms of plumbism. The milder types of lead poisoning are the more common manifestations of the disease in our time, in the adult, and, because of this, much attention has been focused upon biochemical evidences of disturbances in the rates of destruction and replacement of the erythrocytes and the degradation and synthesis of hemoglobin. These disturbances, while not confined to the effects of lead, are among the earlier and more subtle changes induced by lead, which may give warning of more serious symptomatic and organic effects to follow. Even these subtle chemical changes do not occur in association with the levels of the concentration of lead that are prevalent in the tissues, body fluids, and excreta of persons in the general population that have not been subjected to unusual types of exposure to lead. The evidence on this point is extensive and convincing (8, 9, 10, 11); and such evidence, obtained from unusual incidents and accidents within the general population, including those involving infants and small children, and from a wealth of systematic observation of groups of persons engaged in occupations that yield cases of lead poisoning, as well as those that do not, has demonstrated that there is a critical concentration (or a narrow range of concentration) of lead in the body (in the tissues, including the blood, and in the urine), below which none of the obvious or even suggestive manifestations of plumbism have been found. In the intact living body of man, the most readily available tissue for sampling is the blood. This tissue is the most precise indicator, under certain necessary and appropriate conditions, of the lead content of the entire living body and, specifically, of the soft tissues of the body. (The measurement of the rate of the excretion of lead in the urine can often but not always be counted on to yield the same type of information, but the difficulties of sampling and the physiological variability of the excretory process combine to make this measurement more difficult of interpretation than that of the concentration of lead in the blood.)

THE MARGIN BETWEEN THE CURRENT LEVELS OF THE CONCENTRATION OF LEAD IN THE GENERAL POPULATION AND THOSE ASSOCIATED WITH THE OCCURRENCE OF LEAD POISONING

The large proportion of cases of lead poisoning is associated with the absorption of relatively large quantities of lead. The quantities found in individual organs, the blood, and the urine, in typical lead poisoning, are many times larger than those found in the corresponding organs, blood, and urine of persons in the general population. The skeleton, for example, may contain from ten to twenty times the usual levels found in persons in the general population. The liver, kidneys, and brain, likewise, may contain eight to ten times the usual quantities, while the concentration of lead in the blood is similarly elevated, the values ranging as a rule from 0.1 to 0.8 mg. (150 to 800 micrograms) per 100 grams of whole blood, as contrasted with the highest value, 0.05 (50 micrograms) per 100 grams, found among persons in the population who have been carefully screened to eliminate all occupational factors (5, 8). Occasional cases of lead poisoning are found, usually after an intervening period of freedom from exposure occasioned by a delay of some weeks before the blood has been sampled, in which the concentration of lead in the blood is at the level of 0.08 ±0.005 mg. (80 ±5 micrograms) per 100 grams. The latter is the lowest concentration that has occurred, in the experience of the Kettering Laboratory, in association with the onset of lead poisoning in a child or adult, i.e., without an interval of freedom from exposure of more than a few days. This value is not, as some ill-informed persons have believed, the critical level above which one may properly conclude that the person concerned is suffering from lead poisoning. It is rather, as stated above, the level below which no case of poisoning induced by inorganic lead, however mild, has been found in our nearly thirty years of extensive experience with all types of lead poisoning, during which the current, sensitive methods of analysis have been employed. It is not certain, in the absolute sense, that this is the actual threshold level between safety and danger, for it may be (and is believed by certain other investigators to be) slightly in error on the low side. It is also not beyond the bare possibility, in view of the extent of the reduction which may occur in the erythrocytic content of the blood (to which the predominant portion of the lead in the blood is bound), that a bona fide case of lead poisoning may be found at the onset of symptoms, at a slightly lower level. This has not occurred in our experience as yet, despite the fact that no correction of the analytical finding has ever been made for a lowered erythrocytic content. (Due allowance must be made for the analytical deviation which is of the order of 0.001 mg. or 1 microgram per 10 grams of whole blood; and for the calculation, which increases it by a factor of 10, in expressing the result in terms of 100 grams of blood.) This error can be and is partially counteracted, when the result obtained is near, above or below, the critical value, by multiple analyses, by enlarging the sample of blood, or by special manipulations.

In the analytical sense, there is a small margin between the upper limit of the concentration of lead in the whole blood of persons in the general population (who have not sustained any unusual exposure to lead), that is 0.05 mg. (50 micrograms) of lead per 100 grams, and the lowermost limit of concentration which, occasionally, may be associated with the onset of (mild) lead intoxication. In the physiological sense however, if the accuracy of the analytical findings can be substantiated, the difference is not slight, for many months of potentially hazardous occupational (or experimental) exposure to lead are required to elevate the concentration of lead in the blood by an increment of 0.02 mg. (20 micrograms). The reason for this lies in the fact that most of the absorbed lead in the process of its distribution in the body goes selectively into the skeleton, leaving only a small proportion (5 to 8 percent) distributed within the soft tissues, including the blood. Thus, a difference of 0.02 mg. (20 micrograms) per 100 grams of whole blood, as has been demonstrated (4), may be indicative of an increase of more than 100 milligrams in the body burden of lead. It is such physiological facts as these, with their background in known analytical variance, that must be understood before one can consider intelligently the quantitative relationships in this field of toxicology.

LEAD POISONING FROM THE INGESTION OF LEAD

Returning now to the assigned topic, namely the conditions under which lead poisoning may be induced as the result of the ingestion of lead. It is possible to describe such conditions in a fairly precise manner. They may be stated in principle, as follows:

1. Lead poisoning may be induced in a short period of time by a high level of regular, daily, oral dosage, and in a longer period of time by a relatively low level of daily, oral dosage.

2. The requirements are that the dose of lead taken regularly, so as to maintain an essentially constant rate of absorption of lead from the alimentary tract, shall be large enough to lead to a progressive accumulation of lead of critical proportions within the lifetime of the individual.

3. The presence of critical quantities of lead in the intact, living body--the potentially hazardous body burden--can be identified by the finding of a critical concentration of lead in the fluid tissue, the blood, and with somewhat lesser precision by the finding of a critical elevation of the rate of the excretion of lead in the urine.

4. The actual onset of lead poisoning cannot be predicted, nor can this specific illness be identified, with certainty, on the basis of any analytical finding in the tissues, body fluids, or excreta. Only the danger of lead poisoning can be recognized by such means. It is a fact, however, that the likelihood of the actual occurrence of lead poisoning in the individual case is greatly augmented if the concentration of lead in the tissues of the body increases rapidly in response to relatively high dosage. It appears from this fact that if lead is absorbed slowly, some mechanism in the body (beyond the distribution of the major portion into the skeleton) inhibits its toxic effects, possibly by binding it as it is absorbed. The failure of such a mechanism is also indicated by the fact that persons with high body burdens are often precipitated into a bout of intoxication by a sudden brief increase in the rate of their absorption of lead.

The foregoing principles can be translated into quantitative terms on the basis of experiments carried out, within the limits of safety, in the Kettering Laboratory, by means of human subjects, and through certain clinical observations made under fortuitous circumstances. After varying periods of preliminary observation of the metabolism of lead as displayed in a series of young, healthy human subjects, lead was administered by mouth in soluble form, in three doses of equal size, taken with the meals. (These experiments have been described in detail, so that only the pertinent facts need be given here (4, 5).) One subject took 3 mg. of lead as the chloride, daily, for 18 weeks, in addition to that which occurred in his diet during that period. Another took 2 mg. in addition to that in his diet, daily, for approximately 2 years; still another took 1 mg. daily for somewhat more than 4 years; while another took 0.3 mg. per day plus the mean quantity of 0.32 mg. taken daily in the diet, over the period of 60 weeks.

There was a progressive increase in the output and concentration of lead in the urine of the first three subjects, in the concentration of lead in their blood, and in the quantities of lead retained in their bodies (the cumulative difference between the intake of lead in the food and beverages plus that administered), and in the output of lead in their feces and urine. With some daily seasonal and incidental variability, the accumulation progressed at essentially constant individual rates throughout the periods of administration, there being no indication of any decline in rate, even after 4 years in the case of one subject.

The output and concentration of lead in the urine of the fourth subject, whose mean daily ingestion of lead amounted to 0.62 mg. (0.3 administered and 0.32 taken with food and beverages), increased slightly (barely significantly in the statistical sense), and there was a difference of about 12 mg. on the side of intake, between the intake and output of lead, at the end of 15 months, a difference too slight to be considered significant but for the consistency of the trend in this direction from month to month. There can be little doubt that the addition of 0.3 mg. of lead per day to that which occurred regularly in the diet of this subject was sufficient to bring about some slight accumulation of lead in his body. On the other hand, the rate of accumulation was insufficient to yield an elevation in the concentration of lead in the blood in the course of 15 months. It is believed that this experiment has gone about as far in the achievement of a positive result as is possible, and it is clear that a long time would be required, probably longer than the life expectation of this subject, to reach a potentially dangerous concentration of lead in the blood. It would seem therefore that the average intake of lead in the food and beverages of the nation should be maintained for the safety of the public to some quantity short of 0.6 mg. per day, as long as the respiratory intake and absorption of lead remains at or near its present level. In view of the experience of the past thirty years in the U.S., during which there has been no increase in the average oral intake (there has been a slight trend toward a decrease), it should not be difficult to maintain a satisfactory margin of safety in this respect.

The other levels of dosage, namely 3, 2 and 1 mg., daily, continued regularly, in addition to the quantity, approximately 0.3 mg. per day in each instance, taken in with food and beverages, can be counted on, at some time, to yield dangerous levels of the concentration of lead in the tissues, body fluids, and excreta. The period of time required to reach the point of danger (not necessarily of illness, but the distinct threat of illness), in each instance, can be arrived at, with sufficient accuracy for present purposes, by extrapolating on the curves yielded by the values in the blood during the respective periods of observation, to the level of 0.08 mg. (80 micrograms) per 100 grams. These periods turn out to be of the order of 8 months, 4 years, and 8 years, under the three conditions.

The shortest period of time, approximately 30 days, which, in our experience, has intervened between the initiation of the ingestion of lead, regularly, and the onset of a typical bout of gastro-enteric plumbism, was associated with an alimentary intake of 10 to 15 milligrams of lead per day. Of particular interest in this connection were circumstances which conclusively fixed the duration of the period of the ingestion, and the quantities of lead taken daily by two adults, one male and one female, and the dates of the onset of intoxication in the two persons. The dosage and the temporal relationships of these two cases were practically identical.

A single oral dose, in sufficient quantity, of an inorganic (soluble) compound of lead, has been known to cause fatal poisoning within 24 hours. This fact, while of some general importance, especially because the resultant intoxication bears little resemblance to lead poisoning of any recognizable type, is otherwise irrelevant for the purposes of this discussion. The lethal dose, while very large (grams), in the one instance in our experience, could not be ascertained. Oral dosages of several hundred milligrams are capable of inducing acute but not fatal illness in the adult.

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UNDER WHAT CIRCUMSTANCES IS DIRECT CONTACT WITH LEAD DANGEROUS?

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I presume that a discussion of the hazard from external contact with lead and lead compounds has been included in the program for completeness. Let me indicate that this material has only the most tangential relationship to the subject under review at these meetings. My remarks will be brief so that we can devote our time to more fruitful areas of discussion.

This presentation will deal with external contact with inorganic lead, external contact with organic lead, and the toxicological aspects of particulate elemental lead deposits in the tissues. From the practical point of view, the last mentioned item means bullets and shot. One doubts that this subject was to be included as "direct contact," but as it has not been mentioned, I shall mention it briefly.

Whether or not bullets or pieces of elemental lead in tissues can give rise to clinical plumbism has long been a subject of discussion. Obviously many persons have carried such foreign materials around in their bodies for years without showing evidence of intoxication. Lead bullets, however, have not been inert in all cases. Machle (1) showed that the magnitude of lead absorption from these tissue deposits can in some instances approximate the absorption shown by men working in the lead trades.

In one of his cases, a 27-year-old man had carried a pistol bullet in the head of his right femur for six or seven years. There were local symptoms and non-specific complaints such as headache and malaise. Assays for lead in biological materials showed 0.20 mg Pb/liter urine and 0.09 mg Pb/100 ml blood. Following removal of the bullet, there was a steady fall in the levels of lead in body fluids. A second case had a pistol bullet in the right wrist, a urine lead level of 0.07 mg/liter, and a blood lead of 0.07 mg/100 ml.

The fifty or so reported cases of plumbism from retained lead projectiles must represent only a small fraction of persons actually carrying around such deposits. Most of the reported cases would not satisfy present day requirements for the diagnosis of lead poisoning. Perhaps there is some correlation between deposition in joints, where the metal is bathed by synovial fluid, and clinical plumbism. But each situation must be judged individually. Fortunately, if removal of a bullet is being considered because of toxicological rather than mechanical injury, assay of blood and urine lead levels can provide useful guidance.

I must report a remarkable situation at the other end of the spectrum, that of a man who had carried a 2.5 gram bullet in his brain for forty-eight years and died of generalized arteriosclerosis. So much for bullets.

As for the absorption of inorganic lead compounds by direct contact, there is little evidence that this occurs. In fact, the preponderant evidence is to the contrary. One suspects that many if not all of the so-called cases in this category have been related to the oral ingestion of material which had been applied to the skin. This is most obviously the case among women, especially in the Orient, who have applied dusting powders containing lead compounds to their bodies and those of their children. I suspect it is also the case in boys who rubbed white lead putty into marred golf balls to reclaim them or in actresses who used lead-containing theatrical grease paint. Cases have also been reported from hair dye, although once again it is difficult to rule out ingestion or absorption through damaged skin.

In the course of preparing this material I looked into the process of weighting silk, a most interesting procedure. Silk fibers as produced by the silk worm are coated with sericin, a gelatin-like protein, which is somewhat gummy or tacky. This material is removed after weaving and may be replaced with metallic salts such as those of tin or lead-insoluble lead phosphate. The finished product may contain twenty percent or more lead by weight.

Although hundreds of millions of yards of this material have been marketed without reports of deletereous effects, studies were undertaken years ago to define the toxic effect, if any, of lead-weighted silk. (2) One study showed that prolonged contact of these fabrics with body fluids might permit small amounts of lead-containing particulates to become suspended in such fluids, but that their solubility was negligible. Another study (2) of greater potential interest was based on the examination of four women who had worn especially made underwear and nightgowns of lead-weighted silk over the course of six weeks. As far as could be ascertained, the examinations disclosed no effects which could be attributed to the underwear or nightgowns.

As far as absorption through the skin of organic lead is concerned, there seems to be a great variability between reports. Lead oleate (3) seems to be absorbable through the skin of rats, but the evidence for absorption by humans is negligible. Lead naphthenate (4) added as an inhibitor to extreme pressure lubricants in concentrations of 2 - 10 percent may have produced transcutaneous absorption of lead. A situation has been described in which men used a lubricant of this type in the maintenance of rolls in a rolling mill. These men, fourteen in number, filled lubricant tanks, cleaned filters, and routinely soaked their hands and forearms in the fluid. Nine of the men had blood lead levels above 0.05 mg/100 ml and one level was at 0.1 mg/100 ml. Urine leads approximated 0.1 mg/24 hours and after EDTA testing rose about ten-fold. In this situation, however, it is not possible to exclude respiratory exposure from the thermal breakdown of lubricant constituents on the hot rolls.

Experimental studies with these leaded lubricants consisted of massaging into the forearms of each of three volunteers six ml of lubricant (containing 0.2 g. lead). Samples of blood were drawn at intervals. The reports indicate a rise in blood lead by a factor of two after ten minutes and by a factor of three at one hour. Absorption of this magnitude and rapidity is surprising and to my knowledge has not been confirmed.

The most common lead organic is, of course, tetraethyl lead (TEL). Kehoe showed years ago that TEL applied to the skin of rabbits can under some circumstances result in lethal absorption. He also demonstrated that material absorbed into the skin is resistant to removal by washing and is available for distribution throughout the experimental animal over the course of hours. TEL is rapidly decomposed and is thereafter handled as inorganic water-soluble lead. Two practical points must be made here: 1) TEL in gasoline at the 0.1 percent level is absorbed in such small amounts as to be negligible; and 2) in the history of the TEL manufacturing and motor fuel blending industries there has been no instance of organic lead intoxication solely by direct contact - the respiratory route has always been involved in such cases of TEL intoxication as have occurred.

You will appreciate that I have had an opportunity to examine some interesting literature, most of it historical. It bears no significant relationship to the problem of environmental air pollution, which is the subject of your deliberations. You may proceed with your discussions of oral and respiratory absorption knowing that you are not neglecting a third route of importance.

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PANEL: TOXICOLOGY OF LEAD

Chairman: N. Nelson
Co-Chairman: J. Siegel

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HOW SENSITIVE AND HOW APPROPRIATE ARE OUR CURRENT STANDARDS OF "NORMAL" AND "SAFE" BODY CONTENT OF LEAD

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If this symposium had been convened to consider the effects of Europium or some equally unfamiliar element, it would not have been surprising to have heard a progression of speakers cite the known data, then bemoan its paucity, and urge further toxicological research -animal and human- to aid us in arriving at the best possible basis for setting standards of safe exposure. Instead, however, we are convened to consider lead, an ancient and extremely ubiquitous metal, about which more is perhaps known today than virtually any other toxic substance in the general environment. We have so far listened to a full day of presentations in which a great number of laboratory studies, occupational experiences without parallel in any other area of toxicology, epidemiological studies of the general population extending over a relatively long span of time, and all sorts of medical and physiological miscellany have been cited in support of certain principles which must surely rank as the best established thinking of its kind today. In January, 1965, the U.S. Public Health Service issued its report, "Survey of Lead in the Atmosphere of Three Urban Communities", which in many ways would seem to constitute the answers to the questions posed by the U.S. Public Health Service less than a year later at this meeting--and it is natural to wonder at the reasons for such an action.

The topic assigned to me would seem to be the logical concern of this entire gathering. If I could answer the questions posed unequivocally, we could well adjourn--but, of course, I cannot, and I doubt that we collectively shall be able to do so tomorrow morning. Nevertheless, it is well to see what can intelligently be offered in response to the questions posed by the title.

There are four key words in the title which require definition, and agreement thereon by all, before the discussion can proceed. These are "sensitive", "appropriate", "normal", and "safe". "Sensitive" I presume must have reference to our ability to detect minimal damage of any kind, and should be strongly influenced by considerations of the less strong or less healthy members of our general population when applied to air pollution. "Appropriate" must take into account the many practical, perhaps economic, factors which could tend to modify the standards based purely on "sensitivity". "Normal" is a classical trouble-maker, and many writers acknowledge the conflict in definitions by placing quotation marks around the word each time "normal" values are cited; but most of us refer to normal values for any variable as deriving from individuals not known to be exposed occupationally to lead or other substance of interest. Perhaps it would be more correct to define normal values as deriving from subjects whose environment is described or is describable in a certain manner consistent with the purpose of citing such values. It does not seem to me that our ordinary usage of this word implies that it is invariably synonymous with "no effect" level, or "no adverse effect level." Although in the case of lead these may well be synonymous, it is not difficult to cite examples where normal levels are definitely excessive, e.g. natural fluoride concentrations in waters of certain areas.

"Safe," the last of the four key words, is the least definable of all. We all know in a general way what we mean by safe, but many would disagree sharply on the appropriate values for safe levels of lead and other substances. At least part of the disagreement would involve the selection of criteria for defining safe and unsafe conditions. It is in this regard that disagreement is greatest, for some degree of unanimity can be achieved in regard to the meaning of other terms, but to agree on "safe" levels in the air or food, water, body, blood, urine, etc., it is necessary to resolve certain conflicting viewpoints which may not always stem from undisputable objective data. Let me illustrate some of these points by citing certain data which are rather well known, but which must be considered by us in the light of the subject of this discussion.

The title refers to "body content of lead", but for obvious reasons we don't usually measure body content directly, but instead resort to blood, urine, feces, and occasionally other samples of biological origin. Nevertheless, a substantial number of tissue and organ analyses have been performed on samples obtained at autopsy. On the basis of such analyses, as well as calculations of body burden by other means, estimates of body content have been reported by various workers. Several references are cited in Figure 1.

The agreement between the several references cited in Figure I is rather good, so there is little argument over the current body burden of lead, but in contrast to these levels, Patterson (2) has estimated the "natural" body burden for a 70 Kg man in an uncontaminated environment to be about 2 mg, a value which cannot readily be confirmed or denied.

Some notion of the manner in which lead is distributed in the body is given in Figures II and III, wherein typical organ analyses are presented.

As Dr. Kehoe explained yesterday and many times in numerous publications, the concentration of lead in blood and urine relates directly to exposure and body content, and we are, therefore, much more interested in values for these fluids than we are in the actual body content or tissue content, at least when considering living persons. A brief review of some of the significant values is pertinent, although by now most persons in this audience are thoroughly familiar with such values, and redundancy is unavoidable. Figures IV through VII present some of the many interpretations of blood and urine levels which have been made.

Most recently, in the U.S. Public Health Service "Tri-Cities Study" (12) a large number of blood and urine analyses confirm most published data, with only a few values in more than 2000 attaining the upper limits of normal (0.06 mg/100g blood) and (0.07 mg/liter urine). Patterson (2) quotes numerous references and concludes that the present-day blood average is 0.025 mg/100g, with a range of 0.005 to 0.04 mg/100g. These levels he compares to the calculated "natural" level of 0.0002 mg/100g, a value derived in the same manner as the body burden previously cited.

A great many other references could be cited, and in fact, Dr. Stopps of the DuPont Corp. has prepared an extensive review of blood and urinary lead levels in normal populations which presumably will become part of the proceedings of this Symposium, and which confirm in a general way the values cited. One major collection of information which has not been published, and which would be very valuable in attaining statistical validity of a very high order, is the tremendous number of blood lead analyses performed by the automotive industry during past years. In 1965 alone, to my knowledge, the industry will have analyzed more than 40,000 samples. I would imagine that large numbers of analyses have been performed for more than two decades. Surely many of these analyses must be pre-employment samples, perfectly suited to establishing normal levels of the population involved.

Although this discussion is limited to lead levels as such, it should be mentioned that other criteria of lead burden are of interest, particularly when it is believed that lead-induced damage or incipient damage is being measured. Reference to several is included in Figures V and VI, and much has been written concerning some of the urine or blood measurements which can be made. Perhaps one of the more attractive measures of early change which is currently of interest is the presence of delta-aminolevulinic acid in urine, although in a Lead Poisoning Screening Test Study (13) published by the U.S. Public Health Service in 1963, and based on a study of nearly 1,000 children aged 18 to 36 months, it is concluded that this test and coproporphyrin levels cannot be relied upon for screening purposes to predict blood-lead concentrations.

All of the numbers so briefly outlined thus far must be considered in relation to several points where disagreement appears to exist. These are:

1. Although surprisingly good agreement exists on prevailing levels of lead in the body, the concept of "natural" levels in man's early environment must be considered.
2. If near "O" or natural levels are clearly non-injurious, and certain other levels e.g. blood levels of 0.08 mg/100g are believed to be the thresholds of intoxication, does it logically follow that 0.025 average levels, or 0.04-0.05 mg/100g etc., imply intermediate health states wherein a large population at risk, numbering many millions, could be expected to be adversely affected in a manner which would not be noted by an examining physician thoroughly familiar with occupational lead poisoning?
3. Patterson cites the position of the USSR, where the occupational TLV has been set at 10 $\mu g/m^3$, while the 24-hour air pollution limit is 0.7 $\mu g/m^3$. He notes that many people in our cities may be exposed to more than 10 $\mu g/m^3$ and cites the Russian evidence (14) that such levels lead to serious consequences in experimental animals. On the strength of these charges, it seems apparent that there is need for clear cut evidence that the Russian criteria are sound or unsound, and the need for a repetition of some of the animal studies seems evident.

In summarizing my answer to the questions posed by the title of this paper I believe:

A. Our knowledge of normal values of lead in man, aside from semantic difficulties, is virtually unequalled for any other substance and must be considered sensitive and appropriate for virtually any purpose.
B. Our knowledge of the sensitivity and appropriateness of "safe" body levels has been shown to be entirely adequate in the prevention of diagnosable lead poisoning in industry, and it is widely believed to be applicable to the population-at-large.
C. The latter belief has been challenged; it would seem to be the primary responsibility of the challengers to make every effort to describe the alleged adverse effects, and to demonstrate them experimentally.
D. It would likewise seem wise for those who have been challenged to continue their efforts to detect any evidence which argues for the existence of "severe chronic lead insult."

It is obvious that in regard to the effects of lead or any other substance on the health of man, two distinctly different viewpoints may be adopted. It can be argued first that the enormous amount of information presently extant precludes the likelihood of a health state properly described as "severe chronic lead insult;" or the reverse position can be taken, supported by the contention that we have not looked for more subtle manifestations of damage than are currently embodied in the conventional descriptions of lead poisoning. It is dangerous to adopt an "either/or" attitude in such a matter as this, for the complexity of the environment, and the much greater complexity of the effects of the environment on man necessarily lead to complex explanations rather than the more attractive simple ones which are readily understandable.

Lead is but one of many metals present in the atmosphere and diet of man. Further it is only a minor component in the tremendous mixture of chemical and biological matter which permeates the environment. There are many substances present in this mixture which man never encountered at all until relatively recent times, while in the case of many other substances, the quantities which man breathes or ingests have increased sharply since he existed in a "so-called natural" state. It seems unwise to assume that one of these substances can bring about the far-reaching consequences of which lead is accused, without due regard for possible interactions of many substances as well as the ability of the biological system to accomodate itself to a changing environment. This attitude certainly does not rule out the need for continuing research, but it does make it imperative that insofar as possible, the impact of the stress imposed by the total environment be considered.

FIGURE I

BODY CONTENT OF LEAD

Kehoe, Harben Lectures (1)	
Whole body (80 Kg wt)	100-400 mg Pb
Patterson (2), several references cited for 70 Kg adult	
Tipton (3)	90 mg Pb
Krause (4)	220 mg Pb
Monier-Williams (5)	750 mg Pb
Patterson (2)	200 mg Pb (probable average)
Horiuchi and Ida (6)	78 mg (adolescence)
	131 mg (old age)

FIGURE II

LEAD CONCENTRATIONS IN TISSUES

Kehoe, Harben Lecture (1)	mean Pb mg/100g	Other Sources
Brain	0.04	
Lung	0.02	
Heart	0.04	
Liver	0.12	0.19 (Horiuchi (6) 0.17 (Tompsett (7)
Spleen	0.03	
Kidney	0.05	
Muscle	0.03	
Flat Bone	0.65	0.215-1.24 (Horiuchi (6)
Long Bone	1.78	0.311-2.98 (Horiuchi (6)

FIGURE III

LEAD CONCENTRATIONS
PPM IN ASH*

	USA	Africa	Near East	Far East
Aorta	170	76	140	92
Brain	5	5	23	11
Heart	5	5	21	21
Kidney	110	37	70	84
Liver	150	60	77	110
Lung	51	26	47	48
Pancreas	56	17	33	42
Spleen	30	22	58	41
Testis	12	29	28	33

*Tipton (8)

FIGURE IV*

NORMAL VALUES ARE:

Urine (spot sample):
0.01-0.12 mg/liter

(sample of large volume):
0.01-0.08 mg/liter

Blood:

0.01-0.06 mg/100 gms.

(The blood content is the more significant index of absorption, with levels in excess of 0.08 mg Pb/100 gms of blood (not less than two results obtained by methods having a precision of ± 10 per cent) indicative of excessive absorption and the need for cessation of further exposure. When the blood level approaches 0.08 mg per 100 gms, the employee's exposure should be reduced.)

*AIHA Hygienic Guide Series (9)

FIGURE V*

INCREASED LEAD ABSORPTION
(HEALTHY LEAD WORKER)

Blood Lead	<80 μg/100 ml
Urinary Lead	<150 μg/litre
Haemoglobin	>13 g (90%)
Punctate Basophil	$< 10,000/10^6$
Coproporphyrin	+++ (500 μg/litre)

FEELS WELL

*(Lane (10)

FIGURE VI*

HIGH LEAD ABSORPTION
DANGER AREA FOR LEAD WORKERS

Blood Lead	$> 80 \mu g/100$ ml
Urinary Lead	$> 200 \mu g$/litre
Haemoglobin	< 13g (90%) or falling Hb
Punctate Basophil	5-$20,000/10^6$
Coproporphyrin	++++ ($800 \mu g$/litre)

FEELS WELL. OFTEN PALLOR

*Lane (10)

FIGURE VII*

INTERPRETATION OF BIOLOGICAL LEAD SAMPLES

Urine Lead mg/litre		Blood Lead mg/100 gm.	
0.01 to 0.06	non-occupational normal lead absorption re-check: 4 to 8 weeks	0.02 to 0.04	non-occupational normal lead absorption
0.06 to 0.15	occuaptional normal lead absorption re-check	0.04 to 0.07	occupational normal lead absorption re.check: 4 to 8 weeks
0.15 to 0.20	occupational hazardous lead absorption re-check: 2 to 4 weeks	0.07 to 0.08	occupational hazardous lead absorption re-check: 4 to 8 weeks
0.20 plus	occupational absorption compatible with lead intoxication recommend blood lead test, hemoglobin and erythrocyte count, routine urinalysis physical examination re-check: 1 to 4 weeks	0.08 plus	occupational absorption compatible with lead intoxication, recommend immediate re-check and removal from lead exposure area and physical examination

*(11) State of Michigan

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LEAD

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This is a welcome opportunity to review with you certain available data and unanswered questions in the study of lead effect from the viewpoint, which is mine, of a physician interested in clinical preventive medicine in general and in specific diagnostic and therapeutic problems posed by a single patient. Description of my usage of terms in this paper will be useful. The words "toxic effect" connote lead-caused change resulting in no detectable damage or harm. The key word is "detectable." The words "harm," "disability," "damage," here mean that the lead-produced effect cannot be tolerated or handled without producing abnormal function.

It is necessary to emphasize that no harmful effect of lead is unique except perhaps the motor palsy of the most-used muscle group, as in the wrist drop of a right-handed painter whose left wrist is unaffected. This non-specificity of lead intoxication means that identification of low level damage requires a combination of epidemiologic evidence, astute clinical observation to rule out other etiologies together with old and new experimental evidence critically judged for consistency and repeatability.

Another fundamental consideration that weighs heavily in this discussion is the likelihood that lead in the body at levels considered from industrial experience to be harmless can act with other factors to produce damage. If this be true, in judging what amount of lead is harmless for an individual or a population, other insults, natural or man-made, must be assessed.

The biological effects of lead are manifold, exhaustively studied by Aub et al in the 1920's (1). Those of special interest in what I have to present are as follows: first, the action of lead in destroying red blood cells is well recognized and illustrates the two fundamentals just alluded to--the non-specificity of such an effect, i.e., the many causes of such destruction. Further, the hazard to the body of short supply of red blood cells if, for example, there is blood loss from another cause as in bleeding from a peptic ulcer illustrates the problem of more than one factor influencing the pathologic effect. The behavior of lead in following calcium to the skeleton and its mobilization therefrom by such likely events as pregnancy and ageing, is a second, well-established possibility pertinent to this discussion. Third, of potential importance is the accumulation of lead in such organs as the liver and, in the case of organic lead compounds, in the central nervous system. A fourth biological effect, perhaps of the greatest significance, is the damaging influence of lead at certain levels on growth and enzyme activity. I quote from a World Health Organization working paper of Lars Friberg (2) as helpful in introducing this subject:

> "The possibility has been discussed whether certain metals in such minute amounts as are normally present in the body might contribute to the development of chronic disease. The reason for this discussion is the fact that certain essential metals form complexes, for example iron and cobalt in the porphyrin chelates heme and Vitamin B_{12}. Against the background of the laws of chelation it is feasible that other metals might compete for a ligand with an essential metal on a metalloenzyme. The administration of one metal would thus create a condition of deficiency of another. For example, cadmium and mercury would then displace zinc owing to their chelation with ligands which normally chelate with zinc."

Since lead is neither essential nor familiar to the body because it was not abundant in the natural world in which man evolved, it would be surprising if lead did not prove to participate in competition for position in a metalloenzyme.

Experimental evidence that lead may interfere with cellular processes at a molecular level is cited by Westerman, Jensen et al (3) in an article published ten days ago that lead produces mitochondrial morphologic changes and ribosomal abnormalities.

Now to special groups. What I have to offer is a mixture of my critical judgment of old and new data from the literature and my clinical experience. In order to present these data let us agree for the moment that study of large dose effect may be valuable. One can cite benzol as a chemical that can cause aplastic anemia and leukemia after both repeated small doses and a single large dose; the same is true of ionizing radiation. Of the elements, beryllium can result in chronic long-lasting disease, both from an acute dose, causing easily diagnosed beryllium poisoning, and from lower doses, so low that no diagnosable disease is produced at the time.

Borrowing Dr. Alice Hamilton's expression in referring to progeny as offspring, I want to discuss this special group as the first group vulnerable to damaging lead effect. Following is a pertinent quotation from her 1934 edition of INDUSTRIAL TOXICOLOGY (4).

> "There is incontrovertible evidence, both from animal experiments and from observations on human beings, that lead has an injurious action on the germ cells of both sexes and that in addition it passes from the maternal to the fetal blood during the months of gestation (Lewin 349). It is a matter of common knowledge among women who work in lead industries, such as pottery and white-lead production, that lead has an abortifacient action and this action was noted by medical observers more than a century ago. The usually accepted theory as to the mode of action is that lead kills the fetus either by its effect on the germ cell or through the maternal blood and that expulsion of the fetus follows with or without a directly stimulating effect of lead on the uterine musculature (Oliver 474, Kobert 285). Blair Bell (39), however, concludes from his studies on the action of lead on malignant cells, that lead first injures the chorionic epithelium and thus indirectly injures the fetus and leads to its expulsion.
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> "Evidence of the injury to the male germ-cell was first adduced by Constantin Paul (463) in 1860, and a number of French authors have since confirmed his statement as to the abnormal prevalence of abortion and stillbirths in the marital history of male lead workers (Tardieu 584, Ganiayre 158, Deneufbourg 108). Perhaps the most important study in this field is that of Koinuma (306) because it is controlled by comparison with a group of workmen differing from the first group only in the lack of exposure to lead. Koinuma compared the marital life records of workmen, exposed to lead in Japanese storage-battery plants, with the histories of those working in non-lead occupations. The sterile marriages constituted 24.7 percent for the lead group, and only 14.8 percent for the non-lead group; the percentage of pregnancies ending prematurely or in stillbirth was 8.2 for the lead group and 0.2 for the control group."

A passage from Cantarow and Trumper (5) summarized the data on the harmful effect of lead to female reproduction:

> "The passage of lead through the placenta into the fetal tissues has been demonstrated repeatedly.... If born alive, the child frequently shows evidence of the

deleterious effects of lead exerted during the period of intrauterine development. It is often weak and undersized, and there is a high incidence of epilepsy, muscular spasms, convulsions, and other nervous disturbances, idiocy and imbecility (Biondi; Meillere; Prendergast). According to Rennert, in a village where pottery glazing was done in the homes, a large proportion of children had convulsions and a peculiar form of macrocephaly, with a square-shaped head which developed slowly. About 71 percent of a group of 79 such children in this village had either macrocephaly or convulsions or both, and the mortality was 50 percent. It is interesting. . . . that Hall found that 22 percent of the native women in the smelter village mentioned above were unable to nurse their infants because of insufficient lactation, although no difficulty had been experienced before they were exposed to lead.

"There can be little doubt that exposure of mothers to lead has a damaging effect upon fertility, the course of pregnancy and the development of the fetus."

Most authors who have written on the subject are of the opinion that females suffer harmful effect at lower doses of lead than do males. If this is true it is important, making the child-bearing female a second special group. Bell (6), using lead as therapy for malignancy, reports the minimum toxic dose given intravenously as 40 mgm. of lead for females and 100 mgm. for males. Oliver (7), a great student of lead poisoning, states that women are most liable to develop poisoning between the ages of 18 and 23 years, earlier than men and after shorter exposure. Well-studied series have established the fact that women with lead intoxication have a higher incidence of encephalopathy and a lower incidence of paralysis and colic. Such observations have been made in experimental or therapeutic situations or in industries without industrial hygiene controls. The value to us at this meeting of such observations is to point out what lead may do at levels where its effect can be identified. Let me repeat, experience with harmful agents, both chemical and physical, illustrates the critical fact that delayed effect from many small doses may be qualitatively like the more immediate acute effect of a single or few higher doses received over a shorter period of time.

The special group and the one about which most data are available in discussion of lead poisoning is that of the growing child. Rereading Bell's (6) work on the influence of lead on cell-growth one is struck by the very dilute solutions of lead acetate he found to retard growth. In the range of 1 to 100, to as little as 1 to 100,000, all very young tadpoles were killed, whereas more mature tadpoles survived but suffered retarded growth in the 1 to 100,000 solutions. Bell concludes from these and many more studies that "the arrest of growth in animal organisms is strictly proportional to the capacity for, or power of, growth." He further writes that "lead is not only much more toxic to the young and preadolescent than to the adult and old throughout the vertebrate kingdom but also the effects produced by the metal are general in the young (Bell calls this somatic stunting) and local in the adult." There are a number of studies pursuing this point.

Of great importance for this discussion is the mental retardation that follows not only acute lead encephalopathy but perhaps chronic lead absorption. Here are quotations from the discussion by Byers, a pediatric neurologist, at the Lead Hygiene Conference sponsored by the Lead Industries Association* held in Chicago in 1958 (8).

"Certainly when children die who have clinical pathological lead encephalopathy they die with brain stem compression. They die with slowing respiration, pulse rate and blood pressure, anuria, all the usual evidence of increased cranial pressure.

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*Much credit is due this group for conferences, and distinguished research such as that of AUB--one especially recalls Bowditch's enthusiasm.

"I can't believe that this isn't in some way related to interference with the brain enzyme systems through amounts of lead which may not in themselves be impressive.... I originally got interested in lead because of children who had had lead poisoning and had been sent home from the hospital as cured, who then turned up in my neurological clinic because they were misbehaving in one way or another, or not learning in school. One kid set the schoolroom on fire, another nice little girl danced around on the desk. None of these children had acute fulminating encephalitis. They all had evidence of lead poisoning, like stippled cells, some increase in spinal fluid total protein, GI symptoms, coproporphyrin in the urine.

"The point I am trying to make about these children is that though none of them, at two or three years, had an acute encephalopathy with their acute intoxication, when they reached six or seven they showed evidence of injury. I think that many children get chronic encephalitis from lead, as well as acute.... This group of children deteriorates gradually without ever having had any acute lead encephalitis.... I think that lead does something to the growing brain which is different from what it does to the adult brain."

Dr. Rabe, a pediatric neurologist, and I have just begun a five-year study with N.I.H. support of this problem and are including study of siblings of lead-poisoned children with as careful as possible quantitative assessment of the lead to which these children are exposed. Variation in diagnosis and reporting of childhood lead poisoning from urban communities make it certain that little is known of the total number of cases of acute intoxication and nothing of lead absorption in quantities that may pose a risk to normal growth. Some knowledge is provided by Christian et al in Chicago (10). They report that of 9,853 cases of accidental childhood poisoning between 1959-1961, 429 or 4.7 percent were cases of acute lead poisoning, and of the 85 deaths, 67 or 79 percent were due to lead. Colleagues in Boston report 5 to 25 acute cases per year; Baltimore figures (through the courtesy of Dr. Baetjer (11)--in this city there is an excellent detection program--are double this number, reaching over 60 in some years, averaging 57.

During acute lead poisoning in childhood there is reliable evidence that kidney function is disturbed (12). While the blood sugar remains normal, there is readily detected glycosuria, and renal tubular damage has been further demonstrated by aminoaciduria and phosphaturia. In fatal cases acidophilic inclusion bodies in the kidneys are recognized as a pathologic lead effect. These findings may arise from other causes and identification of the lead exposure as causative is sometimes difficult. While Tepper (13) was unable to confirm the findings in Massachusetts, the Queensland (Australia) (14) experience associating serious renal disease in the fourth decade with childhood plumbism is an important epidemic observation which increases the potential for harm to the lead exposed child--clinical illness, which can be recognized, being delayed.

If, as seems likely from the accumulated and growing evidence, growing tissue is more vulnerable to lead than adult tissue, far smaller doses than those required to produce diagnosable lead poisoning in a healthy adult male worker become important. In addition, because homeostasis is not well established in childhood shifts to pH, variation in intestinal acidity or alkalinity which may dissolve ingested lead mean that absorption, distribution and storage of lead are not as well governed as in later life--added reasons that smaller amounts of lead pose greater risks in children. Growing bone is labile, and as lead follows calcium in and out of the bone, unpredictable amounts of lead are alternately deposited and released from the skeleton. This creates the danger of intoxication if lead ingested or inhaled is added at a time of sudden release of lead from bone.

Because of the dominance of my clinical experience, I have been interested in considering what effect lead absorption without detectable evidence of poisoning might have in the medical events of an otherwise healthy individual. The diagnosis of lead damage is not always easy to make because of the non-specificity of symptoms, signs, laboratory findings, sequelae that might all be related to other etiologies. From this line of thinking I conclude that an apparently well lead worker constitutes a third special group for the purpose of this session. Dr. Kehoe's elegant studies (15) are fundamental to the points I raise. With an unmeasured quantity of lead stored in skeleton and liver, with lesser amounts in other parts of his body, such an individual is in some jeopardy, a point Dr. Kehoe made yesterday, and may not be able to withstand either more lead intake without evident harm or may be in more serious trouble than a worker without such lead burden if he suffers an unrelated medical event such as serious blood loss. To illustrate, in a series of men under his care, Belknap (9) reported the hemoglobin values of 15 lead workers (not ill) as between 9.8 and 13.7 grams with 4 under 12 grams although the lead in urine was not very alarming, ranging from 0.12 mgm/L to 0.39/L. Through the courtesy of Dr. Elkins and Mr. Smith of the Massachusetts Department of Occupational Health I have some pertinent data of similar findings in the storage battery industry in that state. Of 72 workers in Pb-using industries in Massachusetts--chiefly in storage battery manufacturing--the average lead excretion during a 7 to 10-year period of exposure is in the range of 0.17 to .58 mg/L. The urine coproporphyrin values (average) ranged from 0.2 to 8.0. The urinary lead excretion was above normal during this period on all occasions of assays in all cases except one. The coproporphyrin excretion followed a less orderly pattern--above normal from time to time in 15 of 72 cases.

That such data reflect a harmful burden of lead is supported by the December 1965 report of Westerman et al (3). These authors found relatively greater amounts of lead in bone by biopsy than in urine of lead workers, at the time asymptomatic. This same study included the important observation that the red cell survival time of three non-anemic lead workers without clinical complaints was reduced and the reticulocyte count elevated, suggesting accelerated red cell destruction and compensatory increased red blood cell formation.

In my clinical experience, the following stand out: A case of peptic ulcer, not held related to work with lead, proved more difficult to handle, requiring transfusion with minimal bleeding. After years of well-tolerated job-related lead exposure, I have seen a case of severely disabling illness with jaundice and two cases of severe headache and dizziness with electro-encephalographic evidence of toxic encephalopathy changes which reversed. These cases followed temporary brief exposure to higher lead levels. I mention these data to urge that it is necessary to consider the additive effect of lead in and out of industry. Gross, easily seen damaging lead effects are at one end of a scale; industrially acquired lead absorption sufficient to reduce hemoglobin in a healthy male may be described as in the moderate potential risk part of such a scale. I believe that low level, constantly though slightly increasing amounts of lead in the body at the lowest end of this imaginary scale presents the body with problems, acting alone or with other harmful agents whose mechanisms of action and site of action are identical with that of lead. . . . This is an appropriate point at which to tackle the ever present variation in individual response to lead exposure. I found a helpful passage in Cantarow and Trumper, though some of the statements are controversial:

> "Although a similarly wide difference in individual susceptibility exists in uniform groups of experimental animals, it is important that, as pointed out by Minot, there

is a consistent quantitative relationship between the amount of lead and the intensity of its effect upon single tissues, such as blood cells, muscle, intestinal strips, etc., no such variability of response being observed as occurs when the intact organism is exposed to lead. As Minot suggests, the individual difference in susceptibility to lead poisoning is therefore probably due largely to variations in factors which influence the amount of lead to which the tissues are exposed rather than to variations in the degree of resistance of the tissues. These include factors influencing absorption, storage, mobilization and excretion (diet, alcohol, constipation, malnutrition, acute and chronic infections, trauma, renal, vascular, and hepatic disease, vitamin deficiencies, drugs, such as iodides, and acid-forming substances and alkalies). Thus, acute symptoms of plumbism may follow injury (Shufflebotham), pneumonia or influenza (Shie), alcoholism (Oliver; Legg and Goadby), renal disease (Edsall; Oliver), etc. As stated by Aub and his associates, immunity is always relative and may be broken down by several factors which influence the general physical condition. Then, too, local tissue damage may render those tissues more susceptible to the toxic action of lead, e.g., renal, vascular, or hepatic disease and anemia (hematopoietic organs)."

Included in possible lead risk to the healthy U.S. male in a Pb-using industry we have learned that cigarette smokers have slightly elevated blood leads (16). In addition, if he drinks beer, the worker will take in .1 mgm $^{Pb}/L$ of beer--if wine, from .08 to .86 mgm $^{Pb}/L$--a problem more serious for European-born workers than most U.S. workers (17). However, these points are pressed to urge that the problem of control of damaging lead exposure be considered not as that of one isolated source but rather a series in view of the widesrpead presence of lead in nature and its ubiquitous use. What is wanted is to consider the total Pb dose the body is expected to handle without irreversible damage.

While the numbers of individuals known to be suffering with such disease may not be great, assessment of the effect of small doses of lead on patients with certain diseases underlines the necessity of evaluating the interaction of lead with another disease entity of a quite separate etiology. For examples I have chosen two diseases. First, because lead destroys red blood cells it has occurred to me that patients with sickle cell trait (thought to be genetic--inheritance due to numberless years of exposure to the malaria plasmodium) might not be more vulnerable to lead. I have seen one such case but was not able to make certain of the quantity of job-related lead and hence not certain of lead as the reason for the markedly decreased red blood cell count. In June 1964, two well-studied cases were reported--two Sudanese workers with thalassemia suffered severe hemolytic anemia as a direct result of lead exposure (18). Such a lead deserves further study.

Cirrhosis of the liver, both from excess alcohol intake and disturbance in iron excretion, is the second disease. Studies of the metal content of such livers show an excess of lead over that found in livers not so affected. Butt et al (19) have studied this problem and report that one-third of alcoholics with cirrhosis store iron and present a metal pattern similar to that in hemochromatosis. They speculate as follows: "Does lead, in response to iron storage, have an insidious and latent effect on hepatic cells, affecting their ability to excrete iron?" Since alcoholic cirrhosis is not a rare disease, the influence of small doses of lead, acting in combination with iron or another metal, deserves further study.

Finally, is there any evidence that lead accumulation influences life expectation or is responsible for an excess mortality of diseases encountered in adult life? Cantarow and Trumper have collected human data and experimental animal evidence up to 1944, and while the conclusions of available studies vary it does appear that lead accumulates with age. More recently Tipton (20), Schroeder and Balassa (21) have shown that lead in human tissues in the United States increases with advancing years.* Schroeder et al (22) have shown that in a carefully controlled lead-free environment, mice and rats fed lead accumulate the metal. In addition, life span was shortened when the lead in the small animal tissues was in the concentration range currently found in humans. Such observations must be repeated and extended.

Turning to knowledge of human disease, the occurrence of nephrosclerosis related to lead is important. The Australian work of Henderson (1955-1957) (24, 25) is impressive. He delineated a distinctive clinicopathologic renal entity in which increase in lead in bone and/or history of childhood lead poisoning was documented. Older literature is pro and con the point that lead causes renal disease, but the majority of investigators believe that either an acute episode of lead poisoning followed by freedom from work exposure to lead or repeated lower level exposures may be responsible for nephrosclerosis. Most convincing is the work of Lane (26) and his colleagues, known to many of you, reported at the Symposium on Lead held in Cincinnati in 1963 and at the International Congress in Madrid that same year. Briefly, the Manchester group presents data to show that lead workers have an excess mortality from renal disease and cerebrovascular disease and at an earlier age than do other males in the United Kingdom.

Lane (26) makes in his well-chosen words the points I raised earlier, regarding the hazard to the health and longevity of the worker who absorbs lead but is not ill:

> "The man who is a "bit below par", who has become slowly and insidiously poisoned with lead but shows none of these acute manifestations, rarely goes to his doctor. Does this matter? This slightly poisoned man is able year after year to carry on with his work. There is apparently little wrong with him. He complains of nothing and he continues to do his job. I maintain that it does matter, that a substantial proportion of these men are being damaged, and that from this group come those who, later on, show evidence of this damage and in some cases die before they should. I believe that from this group come those cases of chronic nephritis due to lead, as well as a number of cases of cerebral hemorrhage."

Comment:

Let me pull together this review. Nonspecificity of sign and symptom, delayed diagnosable damage because of the body's incredible margin of safety, more than one insult acting like lead or with lead require sophisticated attention to the potential effect of low doses of lead--in much the same manner as low levels of ionizing radiation have been studied since the use of atomic energy for military purpose in 1945.

I have reviewed several groups designated as special for the purposes of this meeting. Let me end with what I, with others at this meeting, see as needed change in concept.

There must be a departure in thinking from the present U.S. attitude that prevention of occupational disease is the only requirement of those responsible for the use of such toxic agents as lead. Prevention of diagnosable Pb poisoning in healthy male workers is important but not enough in our society. There is a wealth of knowledge of the biologic effects of Pb and its compounds at hand. There is no available evidence that lead is useful to the body, and all data support the fact that lead is a heavy metal poison. The body's ability to repair damage, its incredibly large reserve, the non-specificity of its responses, help explain the fact that the questions raised by Professor Patterson (28) in his recent controversial article were not raised earlier.

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*The study of Tipton et al (23) includes 200 cases from 10 United States cities.

What action can reasonably be proposed? Well-designed prospective studies of such special populations as I have briefly described here should help establish how long and at what low levels can those in such identified groups tolerate the extra burden of Pb in air, water, soil, food--very different from quantities of Pb that an average healthy 70 kilo man can handle. Cell culture studies with isotopes of lead or stable lead compounds as they occur in 20th century urban air should give those in authority a feeling for how much toxic Pb effect specific organs can tolerate without measurable damage leading to loss of function. Bolder attempts to study the effect of multiple factors acting with Pb on an organ or system must be made. For example, the effect on iron-deficient young animals and adult animals with a source of chronic blood loss given varying amounts of lead might cast light on some of the clinical points I have made. Extension of studies such as those of Byers (29), Smith (30), and our study of M.I.T., among others, relating mental retardation and psychic disturbances in children with measurable excess lead stores both with and without overt Pb poisoning will be helpful. Certainly the sad populations of hospitals for retarded children provide an opportunity to assess the quantity of stored lead with accurate data on lead intake. As ability to study genetic defects increases, such mentally retarded children can be studied with a view to seeing what role, if any, can be assigned to the insult of man-made lead stores.

Finally, I want to quote from Bradford Hill's (31) Presidential Address at the recent founding of the Section of Occupational Medicine of the Royal Society. The title of his address was "The Environment and Disease: Association or Causation."

> "Our observations reveal an association between two variables, perfectly clear-cut and beyond what we would care to attribute to the play of chance. What aspects of that association should we especially consider before deciding that the most likely interpretation of it is causation?"

Professor Hill then discusses the following aspects: strength of association, consistency, specificity, temporal relationship of the association, dose-response curve, plausibility from the biological standpoint, coherence with generally known facts, experimental evidence, and judgment by analogy (using rubella experience to suggest one could agree that another viral disease in pregnancy might be harmful). Professor Hill ends his address with a section entitled "The Case for Action:"

> "Finally, in passing from association to causation I believe in 'real life' we shall have to consider what flows from that decision. On scientific grounds we should do no such thing. The evidence is there to be judged on its merits and the judgment (in that sense) should be utterly independent of what hangs upon it--or who hangs because of it. But in another and more practical sense we may surely ask what is involved in our decision.
>
> "On fair evidence we might take action on what appears to be an occupational hazard, e.g. we might change from a probably carcinogenic oil to a non-carcinogenic oil in a limited environment and without too much injustice if we are wrong. But we should need very strong evidence before we made people burn a fuel in their homes that they do not like or stop smoking the cigarettes and eating the fats and sugar

that they do like. In asking for very strong evidence I would, however, repeat emphatically that this does not imply crossing every 't' and swords with every critic, before we act.

"All scientific work is incomplete--whether it be observational or experimental. All scientific work is liable to be upset or modified by advancing knowledge. That does not confer upon us a freedom to ignore the knowledge we already have, or to postpone the action that it appears to demand at a given time."

I cannot improve on these quotations as satisfying support of my own view of our obligation in the prevention of man-made lead damage, and I look forward to our afternoon session on Control of Lead Pollution.

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PANEL: CONTROL OF LEAD POLLUTION

Chairman: J. A. Zapp
Co-Chairman: J. A. Maga

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POSSIBILITIES OF REMOVAL OF SOURCES OF LEAD CONTAMINATION IN THE ENVIRONMENT

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You may have noted from the program, as I did, that only a single speaker has been designated to discuss how the lead content of the environment may be reduced--and that within a time span of 30 minutes. Naturally I was pleased that the program arrangers throught I might be able to do so; but I am afraid that a really serious discussion of methods for reducing the lead content of the environment must await a finding that reductions are both desirable and needed, plus a designation of priorities for carrying out such a program. In short, if the members of this symposium find that reductions are needed, I forsee that an additional meeting will be required to consider how best to do it.

You are already well acquainted with the pervasiveness of lead in our environment and how it enters into our daily lives in myriad ways. Therefore, you are aware that it will be possible for me to discuss at length only a few of the many aspects of controlling lead contamination of the environment. I hope that the examples I have chosen will prove of interest because of their importance in our deliberations or because they illustrate important principles that are likely to influence whatever actions may be taken.

For convenience, I have divided my discussion into three parts: 1) a consideration of naturally occurring lead contamination, 2) lead pollution from manufacturing operations, and 3) lead pollution from consumptive uses. The contributions of lead to the environment from each of these categories is different in nature and amount, and the control measures that might be taken are quite different, as we shall see.

1. LEAD CONTAMINATION OF THE NATURAL ENVIRONMENT

You have already heard from Professor Warren, Mrs. Cannon, and others that geological lead is distributed more or less irregularly through the earth's crust and is present in the upper layers of soil. From this source, lead enters water and some of this water eventually flows into the sea to bring lead to marine organisms that feed man. Vegetation picks up small quantities of lead from the unpolluted soil and lead enters the metabolism of man and animals from this source also. Possibly, small amounts of lead become airborne in respirable particle sizes from forest fires, volcanic eruptions, and wind action on the distrurbed (as by agriculture) and undisturbed surface of the earth, and by other natural phenomena not under the control of man.

It seems to be resonably well established at this meeting that the quantities of lead absorbed by man from these sources are vanishingly small and represent a negligible fraction of the present total lead intake. It is fortunate that this is so as there appears to be little that might be done to reduce this degree of natural lead contamination of the environment, and no further thought need be given to this source when considering lead control measures.

2. LEAD POLLUTION FROM THE RECOVERY AND FABRICATION OF LEAD AND LEAD-CONTAINING SUBSTANCES

Here, I make a distinction between "contamination" of the environment from natural phenomena and "pollution", which implies contamination through the activities of man.

It is well-known that world-wide consumption of lead is at present about 3 million tons annually and, as is generally true for the utilization of all raw materials, the U.S. consumes the lion's share, approximately 40 percent. This works out to the astonishing annual consumption figure in the U.S. of approximately 15 lbs. per capita. As lead is an extremely durable material in most of its forms, there must be at least 25 times this annual quantity in general usage at any given time. One might look forward to the lead problem working itself out through complete exhaustion of ore supplies were it not for the fact that much of the lead is recovered and reused, lead-acid batteries and type-metal being two familiar examples.

I believe it is safe to say that all operations associated with mining, smelting, chemical conversion, and fabrication of lead and lead compounds result in the release of lead, in some form, to the environment. This may take the form of lead fume escaping to the air from a primary smelter; lead-pigmented paint scraped off the floor of the paint factory and sent to the local land-fill dump; or soluble lead salts finding their way into the waste water system of a chemical plant engaged in the manufacture of lead compounds. I am not aware that reliable figures to evaluate the amount of lead lost to the environment (land, water, and air) from all these sources are available. Judging from the usual experience of processing industries, a loss of lead during manufacture, or processing, equivalent to 0.1-0.01 percent of total production seems to be about the right order of magnitude. If this is true, it means that between one-quarter million and 2.5 million pounds per year are lost to the environment annually in the U.S. from this source alone.

For the moment, it seems probable (once again from analogy with other processing and fabricating industries) that the release of lead to the environment from these operations is local in nature (even though a very tall discharge stack may bring the effluent lead particles to ground some miles from the source). Theoretical mechanisms for widespread dissemination via large rivers and the atmosphere are recognized but, in practice, most of the lead compounds released to the air from factories are believed to deposit close to the stacks by reason of the high specific gravity of metallic lead and most of its oxides and salts, as well as by the fact that only a fraction (and usually a small fraction) of the total particles released from these sources is small enough to be respirable and hence transported long distances by air. The solubility in cold water of most common lead salts is decidedly limited and this is an important inhibiting factor in the rapid leaching of lead wastes deposited on the land. Even when discharged directly to streams, insoluble lead and lead compounds settle rapidly and some, at least, of the soluble materials combine with ions already present in the water to form insoluble compounds (as lead sulfide from H_2S present in septic streams). It is not my intention to minimize the transport of even small amounts of lead from the sites of recovery and processing to other areas, because I think they are important, but I wish to make the point that almost all of the lead discharged to the environment from these sources tends to deposit and remain in the vicinity of the point of origin. It is for this reason that it is convenient to consider these discharges as a local problem (just as the exposure of employees in the lead handling trades is local in nature) both with respect to spatial and population considerations.

There is ample documentation that local lead exposures from these sources may be sufficiently intense to cause acute and chronic lead poisoning to workers, to residents in the vicinity, and to animals grazing in the area. Primary and secondary lead smelters have figured in a number of such episodes.

Fortunately, the technical means for protecting lead workers and preventing the emission of lead to the environment from industrial establishments is already well known and the relatively high lead concentrations present in factory workrooms and effluent streams (as compared with ambient concentrations) make their decontamination somewhat more productive. Indeed, it is a fundamental concept of environment health engineering that controls may be applied most economically at the contamination source. The existence of an adequate body of knowledge concerning the control of lead, plus a corps of technically qualified practitioners does not, of itself, assure universal and adequate application. All who have been concerned with control, rapidly acquire the knowledge that there is a wide diversity in the rigor with which these controls are applied. I had occasion to spend several days inside a tetraethyl lead-producing plant a few years ago and I was greatly impressed with the enormous efforts I observed that were devoted solely to the confinement of TEL to enclosed manufacturing and storage vessels. I have had many occasions, also, to spend time at secondary recovery plants in which lead was being reclaimed from electrical cable and similar salvage materials without controls of any sort to prevent exposure of workers and the emission to the atmosphere of large quantities of lead oxides and other lead compounds from the recovery furnace off-gases.

The points I wish to make are the following: (a) except for a very few of the major producers and fabricators of lead and lead products, the application of known control methods to prevent pollution of the environment from lead and its compounds is far below what might be accomplished were one to insist on achieving the least possible lead emissions. In fact, when controls are instituted at all, it is generally for the purpose of preventing occupational disease and the grossest sort of out-plant contamination. As specific regulations governing the emission of lead to the environment are largely nonexistent in the U.S. (for the reason that there is, as yet, no rational basis for setting non-occupational air quality standards pertaining to lead) economic considerations alone have guided most manufacturers in their decisions regarding the safeguarding of workers and the environment from all but manifestly excessive levels of lead contamination. It seems unlikely that economic considerations alone (once the cost of occupational diseases and gross loss of product have been avoided) will be effective in further reducing the loss of lead to the environment. Many a plant production chief has told me that he knows much cheaper ways of manufacturing his product than by recovering it (or one of its constituents) from the lean tail gases or liquid streams leaving his factory. Therefore, improved control of lead emissions to the environment from manufacturing requires stricter regulations, and doubtless this will result in a compensatory increase in the cost of the product. On the other hand, it seems reasonable to believe that the prospect of major financial outlays for reducing lead emissions to the environment will stimulate engineers to devise improved and less costly control methods. At the same time, I think the industries affected might be somewhat comforted if convincing evidence could be assembled to demonstrate to them that greatly decreased levels of lead contamination are required for health reasons as they already know that 150 μg/M^3 of air is believed to be a safe level for their employees and atmospheric lead levels seldom exceed 10 μg/M^3.

Lead emissions to the environment from recovery and processing plants, though largely local in nature, make a variable contribution to ambient or generalized lead levels depending upon the number, size, and types of lead processing plants in relation to the size of the community in which they are sited. It will be obvious that the impact on the local environment of a 100,000 ton lead smelter located in a steep and narrow valley (where it may be the only industry) will be vastly different in nature and magnitude from the effects of the presence of some dozens of lead salvage operations scattered throughout a vast urban complex such as New York City. I believe that a large fraction of the population is sufficiently remote from sites where important lead processing occurs to be less exposed to environmental lead released from these pollution sources than from lead-containing substances and objects which surround them and with which they come into daily contact, largely unknowingly and as a matter of routine.

3. CONSUMPTIVE USES OF LEAD

I think it is important in this connection to remind you of the facts that were brought out by Dr. Stokinger regarding the great decrease in lead exposures to workers that has taken place over the past 30 years. I am of the opinion that the apparent slight average decrease in lead absorption in the general population as measured by blood and urine values that has been reported for this period is misleading. It seems to me incredible that a 3-fold increase in lead consumption would produce no apparent increase in average lead absorption in the general populations. I am of the opinion that the majority of the population is absorbing somewhat more lead while lead workers alone are absorbing a great deal less. Mr. Stopps has presented some tables that show quite clearly a decrease in the range of lead and urine values (as measured by the standard duration) and a general rise in minima. We recognize that the average values for lead in urine and lead in blood, purportedly from the general population, must inevitably include a number who, currently or at an earlier time, are exposed to abnormal quantities of lead, either occupationally or non-occupationally. If these were eliminated from the averages by careful screening, we might discover a sharper distinction between lead intake and physiological indices of lead absorption. Basically, however, we are concerned at this time about individuals who may respond abnormally to concentrations of lead that are far lower than those we now know to be responsible for clinically recognizable lead intoxications. For this inquiry, evidence concerning the rise and fall of blood and urine average levels in the general population is probably irrelevant.

I wish to speak next about control of lead exposures from non-occupational sources, a matter of much greater complexity and for which, by contrast, a knowledge of suitable and satisfactory engineering controls is at present largely lacking. This is lead pollution from the utilization and consumption of lead and lead-containing substances and objects in generalized usage.

Speaking as an engineer, it is clear that 3 million tons of lead are now being used annually because no other substance, or group of substances can produce equivalent results as cheaply, either by reason of lead's special physical and chemical properties or its relatively great abundance. If it were only a case of substituting a less toxic substance for lead at a slightly greater total cost (as, for example, the further utilization of titanium pigments in place of lead pigments) we might very well be comforted by a conviction that the improvement in the quality of the environment brought about thereby was adequate recompense for the additional monetary outlay. However, if one proposed substituting nickel-cadmium batteries for the familiar lead-acid battery, he would soon find that nickel and cadmium resources were inadequate to fulfill even the present demand for these devices. Therefore, elimination of lead emissions to the environment from the use of lead-acid batteries (which occur almost entirely during manufacture and salvage of lead from used batteries) requires the development of an entirely new substitute, perhaps a thermal-electrical device that permits rapid and direct conversion of heat to current with the burning of a simple fuel such as butane or gasoline. At the present prices, few of us will wish to purchase one of these devices for the family automobile in place of the familiar $20 storage battery. This example illustrates very nicely the miltiple dilemmas we encounter when attempting to eliminate much of the lead in our immediate environment.

It has been demonstrated that the principal modes of lead absorption by the general population in the U.S. are (1) through the respiratory route for airborne lead in the respirable size range, (2) and thus lead ingested with food. Although 90-95 percent of ingested lead is eliminated promptly in the feces, the quantity present in food is sufficiently great to make lead from this source a significant percentage of the total absorbed for those not occupationally exposed.

We have already heard about the many ways that lead enters food during growth and during processing. For example, lead is incorporated into the soil structure from insecticides, from fallout of lead-bearing compounds emitted during lead mining, refining, and fabricating operations, from coal burning, and from automotive exhaust gases, and is metabolized into the plant tissues. In addition, we have heard that lead compounds deposited on plant leaves are consumed by grazing animals and are harvested with the plant for human food. By these means the lead content of plant and animal foods can become markedly increased. During processing in lead containing equipment (e.g. cooking vessels and pipeline containing soldered joints, brass and pewter parts, etc.) and preservation in cans sealed with lead-containing solder, foods pick up additional quantities of lead, although it appears that these sources are rapidly disappearing in the U.S.

It seems reasonable to believe that, if necessary, the lead content of foods could be greatly reduced still further by prohibiting 1) the continued use of lead containing alloys and solders in all food processing equipment, 2) lead-containing seals for cans, and 3) the growing of food crops in the vicinity of major lead producing industries. The lead contribution to food from motor vehicles traversing crop growing regions is not now known but it has been estimated by certain investigators to be substantial. The control of lead emissions from motor vehicles will be considered in a moment. So far as the lead presently in the soil is concerned (it has been estimated to be as high as several thousand parts per million in some industrial areas) it is likely to require a hundred or more years to return to uncontaminated levels by natural means alone.

The health implications of lead emitted to the atmosphere from motor vehicles burning leaded gasoline has its greatest significance for the respiratory uptake by urban populations. About a quarter of a million tons of elemental lead are consumed annually in gasoline additives. Approximately 75 percent, the equivalent of 375 million pounds of lead, is emitted annually into the atmosphere with the engine exhaust gases. As little as 20 percent of the lead burned is emitted during slow driving, the remainder accumulating in the engine and exhaust system. However, during fast driving and rapid acceleration most of the accumulated lead deposit is discharged, with lead emissions rising to 10 or 20 times the content of the gasoline consumed. On the basis of this difference in the emission rate of lead, it has been hypothesized that major releases of lead from automobiles occur "mostly in rural areas" where "high-speed driving conditions ... are encountered." This may have been true a few years ago but now that all our cities are being criss-crossed with limited access highways, much high speed driving occurs inside urban boundaries. For some years now it has been a common experience to drive from one end of Los Angeles to the other at an average speed of 70 mph. Perhaps this explains the significantly higher lead in air concentrations that have been found in Los Angeles compared to other major urban areas.

The complete elimination of lead emissions from motor vehicles may be achieved quite simply by prohibiting additions of lead compounds to fuel. There is precedent for this, although it is not usual or widespread. Other compounds, such as aromatic hydrocarbons, may be substituted to produce the desired anti-knock quality; although it is possible, as claimed by the manufacturers of TEL and TML, that the cost of fuel will rise and the fraction of crude converted to high quality uses will decline, thereby wasting some of our petroleum resources. While this solution to the motor vehicle problem is simple in concept, it entails economic penalties through the increased cost of transportation and the dislocations that would ensue to the producers and users of TEL and related gasoline additives. More drastic solutions, which call for the abandonment of the gasoline eingine and the substitution of Diesel or turbine engines are, at the present time, premature and unattractive. The use of filters to remove particulate lead from the exhaust gases is technically feasible but practically difficult as particles much less than 1 micron in diameter (as you have heard from Mr. Robinson) must be removed at high efficiency without interposing excessive back pressure on the engine.

Before undertaking one or another of these control programs for the elimination or reduction of lead emissions from motor vehicles, it seems desirable first to establish the necessity for their introduction. Important questions concerning the emission of lead from vehicles operating on the highways (as distinguished from test stand vehicles) still remain unanswered. The weight of particles in the respirable size range emitted for various driving situations is poorly defined and virtually nothing is known about their fate after release to the atmosphere except for the data presented at this meeting by Mr. Robinson. One might profitably inquire into the grinding and resuspension of settled lead deposits from vehicular movements; into the oxidation of lead compounds after discharge from the tail pipe; into the agglomeration and settling rates of emitted particles; into the dispersion effects of a rapidly moving vehicle discharging a high temperature aerosol close to road level; and, of course, what changes "clean air modifications" may produce on the patterns of lead emissions from 1966 automobiles.

Similar consideration apply as well to an examination of the need for closer regulation and control of lead emissions from coal burning installations. The size of pulverized coal fly ash varies according to the ash content of the coal and the fineness of grinding prior to firing. A typical Cottrell-precipitated fly ash used by us for many years as a test dust has a mass median diameter of 14 μ, indicating that only a small weight fraction is in the respirable size range. It would be worthwhile, also, to measure the lead content of the respirable fraction of ash particles and to determine what fraction of the lead found in these heat-fused particles is soluble in the lung fluids. If lead emissions to the atmosphere from coal burning prove to be important sources of lead absorption, glass fabric filters may be used to reduce particulate emissions in flue gases to negligible values.

Lead additions to the diet from home storage and preparation of food and drink by way of lead containing utensils and lead glazes on table and cooking ware are believed to be very small at the present time in the U.S. and to represent a negligible addition to the total quantity of absorbed lead because of poor absorption through the alimentary wall. Although ten percent of the annual consumption of lead is for pigments, much less is known about contributions of lead to the air by the weathering of lead-containing surfaces such as paints, glazes, and enameled coatings. In this case, also, it would be administratively simple to prohibit the use of lead-containing surface coatings but the inconveniences, dislocations, and monetary losses that would result, make it advisable to determine that this step is necessary for preserving the well being of the people.

In Summary:

1) Emissions of lead to the environment from mining, smelting, refining, processing, and fabricating lead and lead-containing substances and objects may be greatly reduced, both in-plant and out, by the application of well-known control measures.

2) The remaining principal sources of lead in the environment (lead insecticides, lead-containing surface finishes, lead anti-knock compounds, lead and brass water and waste-water pipes) may be eliminated entirely by prohibiting their further use. Suitable substitutes are available, although they may be more costly and less convenient to use.

3) Additional controls on the introduction of lead into the environment must, in my opinion, be based on a clearly perceived threat to the public health to justify the widespread dislocations and economic hardships that would be certain to follow.

4) Even if it can be demonstrated that present levels of lead in the environment are related causally to a finite number of lead intoxications of some measurable degree of severity, there is no assurance that the population will be willing to make the sacrifices that will be needed to reduce our dependance on lead-bearing products. The non-response of the American people to the Surgeon General's report on the dangers of smoking suggests that it may be exceedingly difficult, if not impossible, to arouse sufficient public action (as opposed to indignant statements) to accomplish anything worthwhile that depends on political action and results in discomforts and hardships to a substantial fraction of the population.

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EVALUATION OF USE VS. NON-USE OF TETRA-ETHYL LEAD IN GASOLINE

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Lead anti-knocks are an essential ingredient in gasoline. Their use conserves natural resources. They are used for economic reasons and thus I wish to direct your attention primarily to an appraisal of economic aspects of the "Evaluation of Use vs. Non-Use of TEL in Gasoline."

First, a word about our refinery technology operations might be in order. Our Refinery Technology activity is an engineering organization whose principal function is to help petroleum refiners make the most efficient use of anti-knock compounds. Over the years, this group has worked closely in literally hundreds of studies with essentially every gasoline manufacturer in this country and many more throughout the free world on one phase or another of problems related to gasoline manufacture, blending, and use. Our work primarily involves consulting services on the optimum use of anti-knocks. This area, in turn, involves:

1. The most efficient operation of processing equipment to produce gasoline components.

2. The most efficient blending of gasoline components to finished gasoline.

3. The tailoring of the anti-knock quality of gasolines to the octane-number requirement of automobiles.

Because optimum utilization of anti-knock compounds has a direct bearing on all these functions, this study draws on our extensive, thorough, and authoritative background in discussing the economic aspects of the use of lead anti-knock compounds in gasoline.

At this time, I would like to highlight the results of two studies. The first is an appraisal of the effect of elimination of lead from gasoline on the U.S. Petroleum Refining Industry as a whole. The second will be a description of the situation faced by a moderately sized refiner in order to illustrate the nature of problems individual refiners would encounter.

Petroleum refining economics is a complicated subject. For this reason, sound studies of economics must be complex and must use the latest tools of economic analysis. Any simple approach will tend to overlook important considerations. Recognizing this complexity, in both studies we have used the modern management science technique known as linear programming to simulate refinery operations, with solutions obtained through the application of large electronic computers. Today, this approach is recognized as the standard and accepted method for such investigations. Because we have not taken a simple approach, my subject matter is also necessarily fairly complex.

For the industry study, we employed a model that simulates the operation of the entire U.S. refining industry. This model is used by Ethyl to forecast future demand for anti-knock compounds. It effectively reproduces industry activity in historical periods on which statistical data permit a check. Furthermore, we have had extremely good success at prediction of near-future demand with this model. In the lead use vs. non-use study, simulated situations evaluate the effects of producing gasoline without lead anti-knocks in contrast to a base case that reflects present-day operations with lead. We compare least-cost situations with lead to least-cost situations without lead so that each way of operating is fairly represented.

This Table 1 presents for the U.S. refining industry raw material intake and product distribution for the base case using lead and a case simulating how the industry would be forced to operate if lead anti-knocks were unavailable. Raw material prices and product values assumed for this study also are shown in this table. For the leaded base case, the volumes of raw material intake and the volume of products heavier than butane are consistent with published statistics for 1963. Statistical data for 1963 are used in this study since this is the latest annual period for which complete information was available at the time work was started.

The "no-lead" case makes the same volume as the leaded case of all products heavier than butane. This is the only reasonable assumption that can be made since the industry would have to satisfy the demand for all products whether lead anti-knocks were available or not.

It has been assumed for simplicity that aviation gasoline can be made in the "no lead" situation. Actually, this assumuption is not really valid since high-quality aviation gasoline cannot be made if lead anti-knocks are unavailable.

Excess hydrogen, fuel gas, and propane are valued as fuel in both the lead case and the simulated "no lead" case. Butane is valued at 6¢ per gallon in the leaded base, which is consistent with current values. It is valued at fuel price in the case without lead because fuel gases are produced in excess of base volumes and because light hydrocarbons become impractical components for gasoline blending with the type of processing required to manufacture unleaded gasolines.

In the "no lead" case, crude oil and natural gas liquids intake are allowed to vary to permit attempted production of the same quality and quantity of petroleum products as in the leaded base case. As a result, crude oil consumption increases more than one million barrels per day and the consumption of natural gas liquids decreases to zero, with an overall increase in total petroleum raw materials of about 1/2 million barrels per day. Overall, raw material intake increases by more than 5 percent. However, the consumption of crude oil increases by more than 10 percent. We are convinced that the manufacture of gasolines without lead anti-knocks would be very destructive of our petroleum natural resources.

The consumption of natural gas liquids decreases for the same reason that the value of butane must be degraded. The type of processing required to manufacture unleaded gasolines makes the highly volatile natural gas liquids impractical raw materials for petroleum-refining operations. Consequently, the natural gas industry would have to find alternate uses for these materials or they would become low value by-products of natural gas production.

This Table 2 shows processing investment and operating costs for the various types of processing considered available in this study. Investment costs which are expressed as dollars per barrel of capacity, include a charge of 20 percent of processing unit cost for the cost of off-site facilities and can be thought of as average costs to the whole industry or as specific costs to refineries with crude running capacity of roughly 75,000 B/D. Unit, fixed, and incremental costs are all expressed on a calendar-day basis. Fixed costs are equivalent to 36 percent per year of capital investment. Included are charges for increased maintenance, insurance, property taxes, depreciation, interest on borrowed capital, income taxes, and return on investment. No charge for increased working capital has been made in this study to cover such factors as the increased inventory of crude oil, catalyst, and hardware that would be required. No allowance has been made for the increased operating labor, supervision, and technical support of operations that would be needed with the increased complexity of operations.

This Table 3 shows, for the U.S. refining industry, the through-put of material for various petroleum-refining processes. Again, the data for the leaded base case are consistent with the availability of various types of processing facilities in the U.S. refining industry in 1963. For unleaded gasoline, the figures shown represent the capacity required to produce the slate of products demanded of the industry. The next column of figures reflects the new processing required by the industry to make gasolines without lead. No excess capacity is assumed in the no-lead case just as no excess capacity has been assumed for the leaded base case.

The investment in new processing facilities required to permit the U.S. petroleum refining industry to produce gasolines without lead anti-knocks would be about $3 billion. This is roughly 1/3 of the industry's current total investment in petroleum refinery process facilities. Further, it is six or seven times the current high annual level of capital expenditures in new petroleum refining facilities; and current expenditures are straining the capabilities of construction companies. Current expenditures are being used for expansion of manufacturing operations to provide for growth in demand, to improve product quality, and to increase the efficiency and flexibility of current operations. The multibillion dollar investment required to make gasoline without lead would not provide for needed growth, quality, flexibility, and efficiency. In fact, a loss in flexibility, efficiency, and gasoline quality from current levels would be experienced without antiknocks.

This Table 4 shows, for the U.S. refining industry, the properties of gasolines produced in the lead case and in the case with no lead. In the leaded base case, Research and Motor octane numbers for premium and regular gasolines are U.S. average values for 1965. Volatility properties of lead case premium and regular gasolines are average U.S. values for all areas and all seasons of the year and are derived from Bureau of Mines survey data. These properties are Reid vapor pressure and percent of gasoline evaporated at the stated temperatures in degrees Fahrenheit. Hydrocarbon-type analyses for the leaded base-case gasolines are calculated from the composition of gasolines obtained in the simulation of the U.S. refining industry and are representative of today's fuels.

Moving on to the specification column, present-day Research and Motor octane numbers have been used as minimum standards for fuels produced without lead anti-knocks. The volatility specifications for unleaded gasolines have been set so as to provide reasonably satisfactory performance, although somewhat less satisfactory than average leaded gasolines. It is practically impossible to produce gasolines without lead that would have the same volatility characteristics as present-day fuels and at the same time approach current standards of octane quality. Thus, we have assumed U.S. average volatilities could shift somewhat from present averages but that the limits of the current range of gasoline volatilities would be the limits of acceptability and that the U.S. average volatility would have to fall within this range and could not be on either extreme.

Hydrocarbon-type specifications are not imposed on either the lead case or the "no lead" case.

Now let's consider the last column, which shows the gasoline properties for the no-lead case. Experimental and commercial data show that gasolines produced without lead anti-knocks become more sensitive; this is, the differences between Research octane number and Motor octane number increases. Thus, Research octane number must be raised above current levels to maintain current Motor octane number. For premium gasoline made without lead, Research octane number goes from 100.0 to 101.3 to maintain the Motor octane number at 91.2. For regular gasoline, the Research octane number increases from 93.9 to 96.9. Experience and experimental data show that, despite the higher Research octane number, these unleaded fuels are more prone to knock in cars on the road than today's leaded gasolines. A recent study involving ten cars and fuels similar to those shown in this table verifies this conclusion. Thus, gasolines made without lead anti-knocks will satisfy fewer of the cars on the road than leaded gasolines, even if laboratory octane quality is maintained.

Although we have attempted to maintain reasonable control on the volatility quality of gasolines produced without lead, the premium gasoline tends to become heavier. Without some volatility specifications, it would become much more so. On the other hand, the regular grade gasoline becomes more volatile for the no-lead case. Again, this tendency would be more pronounced without reasonable volatility specifications. Directionally, these problems become very severe if a significant part of the required capital investment is not devoted to control of gasoline volatility. But, if we ignored volatility specifications, the resulting premium fuel would be unsatisfactory in warm-up characteristics and would produce higher hydrocarbon emissions. On the other hand, the regular grade gasoline would be unsatisfactory in terms of cold weather carburetor icing and warm weather vapor-lock tolerance, and the evaporative losses would become severe.

It is desirable to have similar volatility in both grades of gasoline - both from civilian and military points of view. Today's premium and regular gasolines have similar volatilities but this practice would become impractical if lead were not available. Again, the hydrocarbon type analyses for the no-lead situation are calculated from the compositions of the gasolines obtained in the simulation study.

This Table 5 summarizes the main conclusions from this part of the study and shows the increased cost of manufacturing gasoline if lead anti-knocks were not available. The cost of making gasolines approaching the quality of today's gasolines without using lead anti-knocks is estimated to be 2.6 ¢/gallon for the U.S. refining industry as a whole. This is a 30 percent increase in the manufactured cost of gasoline (including raw material cost) based on an assumed present-day cost of 8.5¢/gallon for an efficient large refinery. Principal components of the increased cost of gasoline are the increased consumption of petroleum raw materials (185,000,000 barrels/year) and the enormous capital investment (3 billion dollars) in new process facilities that would be required. The type of gasolines which would have to be produced without lead would be less satisfactory than today's fuels and would require serious consideration with regard to certain problem areas.

1. Premium gasolines would become heavier, and thus would have poorer warm-up properties and produce higher hydrocarbon emissions.

2. Regular gasolines would become lighter, and therefore would be prone to carburetor icing, would have less resistance to vapor lock, and would produce higher evaporative losses.

This Table 6 highlights three other areas that should be considered. Reduced flexibility of refining operations is a serious problem that would face the industry if anti-knock compounds were unavailable for use in gasoline blending. This loss of flexibility manifests itself in two ways:

1. It would reduce the industry's current capacity for providing emergency supplies of gasolines, jet fuels, and specialties on short notice. Refiners would no longer be able to incorporate large volumes of marginal gasoline stocks or release potential high-octane gasoline feedstocks or high-octane components for other uses.

2. It would complicate gasoline blending. Increased time and effort would be required to blend gasoline from components. Inventories of components would have to be larger. This loss in efficiency has been estimated to require an increase of at least one-half number for all octane targets to assure satisfying specifications.

It has been suggested by a few individuals that, rather than restore present octane quality with processing, cars should be modified to operate on the quality of gasoline that could be produced by simply leaving lead anti-knocks out of gasoline. Assuming that present-day cars could be made to perform on fuels of that quality as well as they perform on present fuels (and this assumption is not valid), fuel economy would be adversely affected. An estimated 10 percent increase in gasoline consumption would result. This increased gasoline consumption would cost the motorist an additional 2 billion dollars per year based on a gasoline price of 30¢/gallon as well as depleting crude oil resources and increasing total hydrocarbon emissions proportionately.

Still another factor should be considered. If forced to operate without anti-knocks, the refining industry would be dependent to an even greater degree on the availability of platinum catalyst for catalytic reforming as a means of producing needed octane quality, both in normal times and in times of emergency. Since platinum is not a native raw material, the United States is subject to the availability of this metal in the world market. The principal world source of platinum is the Soviet Union, with South Africa a secondary source. Very small amounts are produced in the Western Hemisphere.

We estimate that the present capacity for catalytic reforming would have to be essentially doubled if gasolines had to be manufactured without lead. This means that the industry's inventory of platinum for reforming catalyst would have to be doubled. Furthermore, the future consumption of platinum would more than double due to the increased severity of processing. Roughly, 1/2 million troy ounces of platinum metal would be required to provide the initial catalyst charge for new catalytic reformers. This is about equivalent to the total annual U.S. consumption of platinum and four times the 1964 petroleum industry consumption which was considered spectacular by the Bureau of Mines, according to an Oil & Gas Journal article.

All discussion to this point has related to the U.S. petroleum refining industry as a whole. Thus, we have been looking at the average situation. Actually, there is no average refinery, and each refinery in the country would encounter unique problems in attempting to make gasolines without lead. To illustrate some of these "non-average" problems, brief comment on a specific refining situation is needed.

Let's consider the case of a moderate-sized Midwestern or Eastern Great Lakes refiner located near a major city. This refinery has a crude running capacity of about 20,000 B/D. There are about 65 gasoline manufacturing refineries in the U.S. that are the same size or smaller than the one that will be illustrated. Thus, this is not an extreme case. The smaller refineries would be less capable than our 20,000 B/D refiner in dealing with the problems of manufacturing gasoline without lead.

This Figure 1 shows a simplified flow diagram of our refinery. It contains the essential types of processes needed by refiners to competitively produce quality products for today's market with lead anti-knocks available.

Our illustrative refinery differs from larger refineries in many ways, but a few differences are of greatest importance to the subject of anti-knocks. Particularly, this refiner differs from the average in the following ways:

1. Higher average octane quality on both premium and regular gasoline.

2. Slightly higher percentage of premium gasoline on total gasoline.

3. Higher percentage of gasoline on crude.

4. Smaller size processing units, resulting in higher capital costs in $/barrel of capacity, less flexible operations, and limited sizes of economically justified unit operations.

The current investment in process facilities for this refinery is about 20 million dollars.

In this Figure 2 the additional processing units required to manufacture gasolines without lead anti-knocks are shown. The required investment in new processing facilities totals 13-1/2 million dollars or about 2/3 of the total current investment.

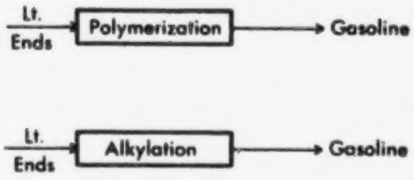
At the present time, the management at a refinery of this size could well be seriously considering the relative advantages and disadvantages of adding a hydrocracker to the present processing scheme. In such consideration, a major concern would be whether the "1 million dollar" investment can be justified in terms of improved product distribution or quality or even the banker's willingness to finance such an investment. For this refiner, contemplating a $13-1/2 million investment in new facilities seems impossible with no improvement in produce quality, or product slate, or operating efficiency.

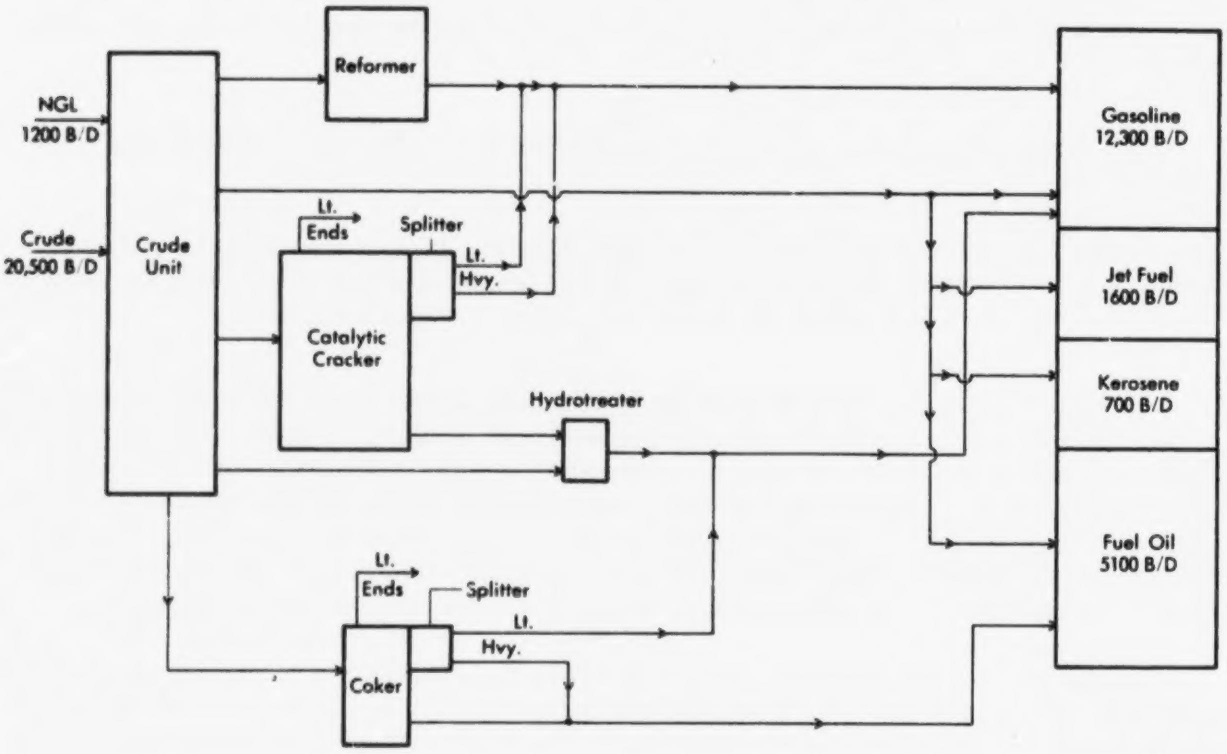
The problem areas noted for the U.S. refining industry apply for this illustrative refinery as well, but become more severe. Premium gasoline would become heavier, and regular gasoline would become more volatile. These properties would be harder for the small refiner to hold in line. Petroleum raw material intake would increase about 10 percent and the manufactured cost of gasoline would increase almost 4 ¢/gallon, or about 45 percent.

Assuming that the cost of manufacturing unleaded gasolines could be passed from the refiner to the motorist, injustices would inevitably be created. Refiners with costs lower than the average would benefit whereas refiners with costs above the average (such as the one illustrated) would suffer. Directionally, the small refiner, whose costs would almost always exceed the average, would be hurt the most. At least 100 small refineries (roughly 1/3 of the total number of refineries in the United States) would probably have to be closed. We have been told by small independent refiners that they would be unable to compete in the gasoline manufacturing business if anti-knocks were unavailable to them as a means of producing competitive anti-knock quality. These refiners would be forced out of the petroleum refining business.

FIGURE 1

REFINERY USING LEAD

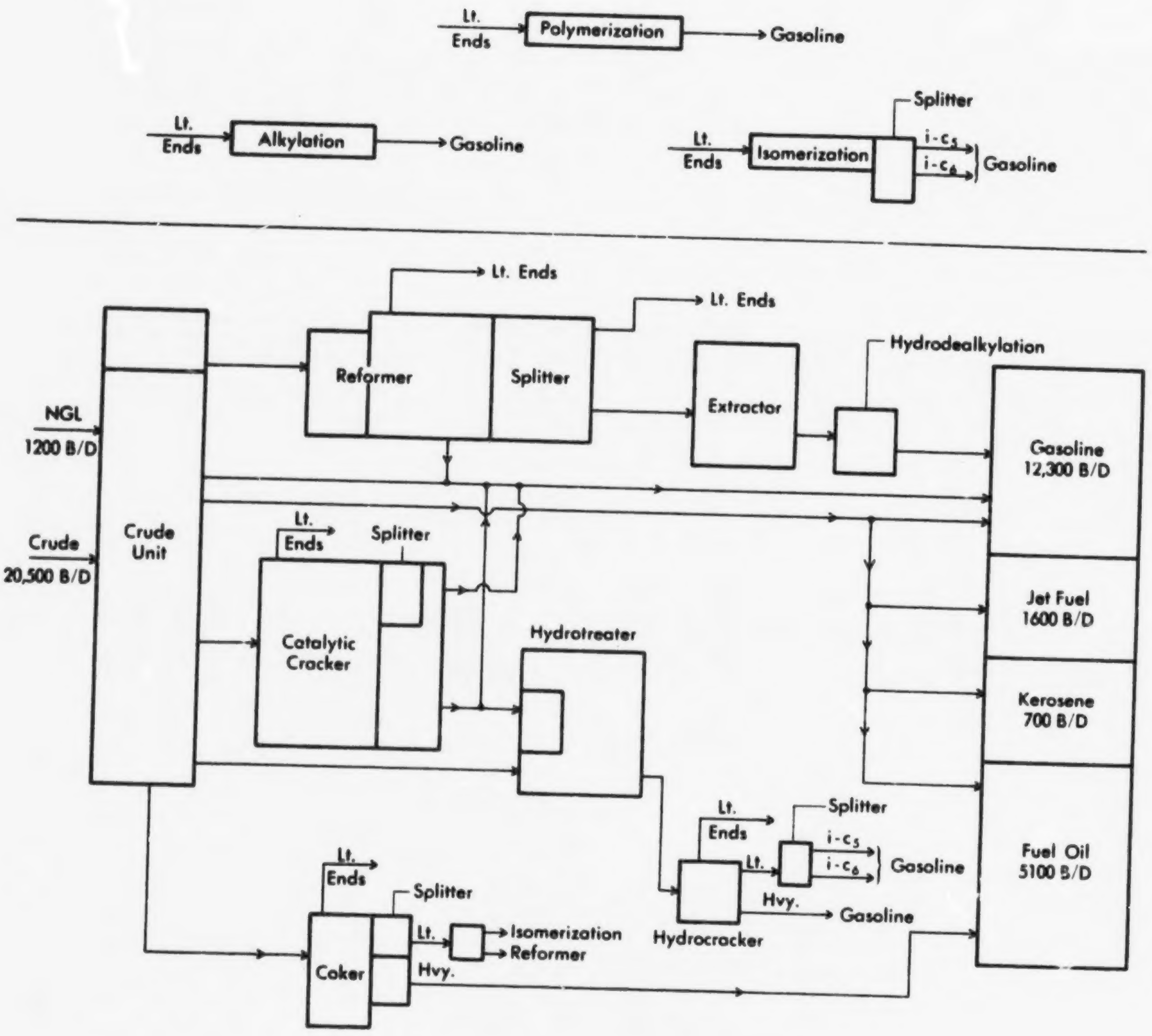

Lt.
Ends
Polymerization
Gasoline
Lt.
Ends
Alkylation
Gasoline


NGL
1200 B/D
Crude
20,500 B/D
Crude Unit
Reformer
Lt.
Ends
Splitter
Lt.
Hvy.
Catalytic Cracker
Hydrotreater
Lt.
Ends
Splitter
Lt.
Hvy.
Coker
Gasoline
12,300 B/D
Jet Fuel
1600 B/D
Kerosene
700 B/D
Fuel Oil
5100 B/D

Represents $20,000,000 Investment

FIGURE 2

REFINERY USING NO LEAD


Lt. Ends
Polymerization
Gasoline
Lt. Ends
Alkylation
Gasoline
Splitter
Lt. Ends
Isomerization
i-c5
i-c6
Gasoline
Lt. Ends
Lt. Ends
Reformer
Splitter
Hydrodealkylation
Extractor
NGL
1200 B/D
Crude
20,500 B/D
Crude Unit
Gasoline
12,300 B/D
Lt. Ends
Splitter
Catalytic Cracker
Hydrotreater
Jet Fuel
1600 B/D
Kerosene
700 B/D
Lt. Ends
Splitter
i-c5
i-c6
Gasoline
Lt.
Hvy.
Gasoline
Hydrocracker
Fuel Oil
5100 B/D
Lt. Ends
Splitter
Coker
Lt.
Hvy.
Isomerization
Reformer

Represents Additional $13,500,000 Investment

Table 1

U. S. REFINING INDUSTRY INTAKE AND PRODUCTION

Intake	Raw Material Price $/B	Leaded Base M B/D	No Lead M B/D	Increase for No Lead Case M B/D
Crude Oil	3.05	8,717	9,800	1,083
Natural Gas Liquids	2.31	579	--	-579
Unfinished Oils	--	87	87	--
Total Petroleum Raw Material		9,383	9,887	504
Production				
Excess H_2. M SCF	10 ¢/MSCF	763	497	-266
Fuel Gas, FOE - 30.5 ¢/$\overline{M}$ Btu	1.92	339	581	242
Propane - 30.5 ¢/$\overline{M}$ Btu	1.17	268	585	317
Butane - 6 ¢/gal.	2.52	139	442*	303
Premium Gasoline @ 12.3 ¢/gal.	5.15	1,409	1,409	--
Regular Gasoline @ 10.5 ¢/gal.	4.41	2,835	2,835	--
Aviation Gasoline @ 13.0 ¢/gal.	5.46	146	146	--
Naphtha Specialties	6.30	62	62	--
Petrochemicals	6.68	113	113	--
Military Jet Fuel	3.89	226	226	--
Kerosene & Comm. Jet Fuel	3.57	495	495	--
Diesel Fuel	3.36	628	628	--
Heating Oil	3.30	1,466	1,466	--
Residuals & Road Oils	2.05	777	777	--
Lubricants	10.29	173	173	--
Asphalt & Wax	2.55	321	321	--
Coke, Tons	12.00	89	89	--
Miscellaneous	8.40	99	99	--

* Valued @ 30.5 ¢/$\overline{M}$ Btu

Table 2

U. S. REFINING INDUSTRY PROCESSING INVESTMENT & OPERATING COST

Processing Equipment	Investment Costs* $/B of Capacity	Operating Cost, $/B Fixed**	Operating Cost, $/B Incremental
Cat Cracking	400	0. 3944	0. 10-0. 12
Hydrocracking	720	0. 7099	0. 36-0. 38
Alkylation	1, 017	1. 0027	0. 55-0. 99
Isomerization, C_5 - C_6	440	0. 4338	0. 30
Cat Reforming	400	0. 3943	0. 30
Hydrotreater	200	0. 1972	0. 19-0. 48
Extraction	467	0. 4605	0. 05
Fractionation	26-67	0. 0256-0. 0660	0. 20
Hydrodealkylation	1, 071	1. 0560	0. 02
Crude Unit	100	0. 0986	0. 69
Hexane Isomerization	512	0. 5048	0. 10
BTX Facilities	--	--	0. 30
Polymer Plant	--	--	0. 25
Thermal Processing	--	--	0. 45
Coker	--	--	--
Cracker	--	--	0. 11
Visbreaker	--	--	0. 10-0. 20
Alkylation Revamp	165	--	0. 05
Cat Reforming Revamp	50	--	--

* Includes 20% of battery-limit installation for off-site.

** 36% of capital investment per year. Includes 4% maintenance, 2% insurance and taxes, 30% depreciation, interest, and return on investment.

Table 3

U. S. REFINING INDUSTRY PROCESSING THROUGHPUTS

	Leaded Base M B/CD	No Lead M B/CD	New Processing Required M B/CD	Investment $/B	Investment MM $
Crude Fractionation	8,717	9,800	1,083	100	108
Catalytic Cracking, Fresh Feed	3,545	3,442	--	400	--
Hydrocracking	38	849	811	720	584
Thermal Processing	1,127	764	--	--	--
Catalytic Reforming	1,688	3,505	1,817	400	727
Extraction	--	1,075	1,075	467	502
Hydrodealkylation	--	113	113	1,072	121
Isomerization, C_5 - C_6	8	958	950	440	418
Isomerization, C_6	--	--	--	512	--
Alkylation	416	447	31	1,017	31
Polymerization	99	59	--	--	--
Hydrotreating	238	663	425	200	85
Fractionation of:					
Cat Cracked Gasoline (Lt/Hvy)	620	1,481	861	26	22
Cat Cracked Gasoline (C_5/C_6/Hvy)	--	--	--	--	--
Thermal Gasoline (Lt/Hvy)	336	148	--	--	--
Reformate (C_5 - C_6/C_{7}+)	--	2,514	2,514	26	65
Isomerate (C_5/C_6)	--	395	395	67	26
Lt. Hydrocracked	--	229	229	67	15
Lt. Thermal	--	86	--	--	--
Alkylation Revamp	--	416	--	165	69
Catalytic Reformer Revamp	--	1,688	--	50	84
					2,857

Table 4

U.S. REFINING INDUSTRY GASOLINE PROPERTIES

	Leaded Base	Specifications	No Lead
Premium			
Research Octane Number	100.0	100.0 min.	101.3
Motor Octane Number	91.2	91.2 min.	91.2
TEL, ml/gallon	2.54	0.0 max.	0.0
Reid Vapor Pressure, psi	10.2	10.2 max.	10.2
% Evaporated @			
158°F	28	25 min.	27
180°F	36	50 max.	34
240°F	62	57 min.	57
356°F	97	90 min.	99
Hydrocarbon Type, Vol %			
Aromatics	22	--	47
Olefins	16	--	--
Saturates	62	--	53
Regular			
Research Octane Number	93.9	93.9 min.	96.9
Motor Octane Number	85.5	85.5 min.	85.5
TEL, ml/gallon	2.05	0.0 max.	0.0
Reid Vapor Pressure, psi	10.2	10.2 max.	8.2
% Evaporated @			
158°F	32	20 min.	32
180°F	43	47 max.	47
240°F	62	50 min.	67
365°F	95	90 min.	100
Hydrocarbon Type, Vol %			
Aromatics	17	--	35
Olefins	20	--	21
Saturates	63	--	44

Table 5

U.S. REFINING INDUSTRY SUMMARY

COST OF MAKING GASOLINE WITHOUT LEAD ANTIKNOCKS

Increased Cost of Manufacturing Gasoline	2.6¢/gal. of gasoline	
Percent Increase in Manufactured Cost	30%	

Increased Petroleum Raw Material Consumption	Barrels/year	%
Crude Oil	396,000,000	12
Natural Gas Liquids	-211,000,000	
Net Increase	185,000,000	5

Investment in New Processing Facilities	$3,000,000,000

PROBLEM AREAS

Premium Gasoline heavier (poorer warm-up and higher hydrocarbon emissions)

Regular Gasoline lighter (more prone to vapor lock and higher evaporative losses)

* * * * *

STATEMENT

C. G. Gerhold,
Executive Assistant to the President,
Universal Oil Products Company,
Des Plaines, Illinois

I would like to preface my remarks by making it very clear at the outset that the Universal Oil Products Company is not making any recommendations with regard to the use of lead in gasoline. Questions as to whether the amounts of lead emitted from automotive exhausts are tolerable or harmful must, we believe, be left to those of you who are more expert in this field. Our comments will deal solely with the technical feasibility and economics of producing a gasoline meeting current quality standards, without the use of lead.

The anti-knock characteristics of gasoline sold by the petroleum refining industry have steadily increased throughout the years. In addition to the adoption and continuing use of lead compounds, oil companies have spent very substantial sums of capital in making use of a series of new refining techniques which improved these characteristics of their gasolines by modifying their basic chemical nature. The first of these was thermal reforming, which was followed by catalytic polymerization, catalytic cracking, alkylation, catalytic reforming, isomerization and, most recently, hydrocracking.

Our company, the Universal Oil Products Company, takes great pride in its important role in creating much of the improved technology which has made these quality improvements economically possible. It maintains a substantial research and development organization which is continually active in this area, and several of the listed processes were based almost entirely on its successful research and development activities. In this connection, you may be aware that one of our scientists was awarded the "Modern Pioneers" medal by the National Association of Manufacturers at its recent national meeting, in recognition of his scientifically significant and commercially important contributions to the technology of refining for octane number improvement.

The development and supply of technology to enable the petroleum refining industry to provide economically the amounts and types of products it needs for its markets have, for the more than fifty years of its corporate life, been the principal business of the Universal Oil Products Company. This technology is supplied to petroleum refiners the world over through process licenses, technical services, and catalysts and additives used in the production of petroleum products.

Since its processes, services, and products must be not only scientifically, but also economically sound, the Universal Oil Products Company has, in the normal course of its business, also become very familiar with all phases of refining economics.

As an essential part of our petroleum-oriented activities, our organization must anticipate trends in the industry's markets to enable it to be ready with new developments at the time they are needed by our refining customers. Several years ago we became disturbed by the growing tendency to attribute a major share of the blame for decreasing air quality to the use of the petroleum industry's products and began to devote considerable attention to the problems which the industry was facing, as a result.

We do not agree with those who feel that the solution to these problems lies in placing limitations on the use of petroleum products and conversion to other sources of energy. Rather, we favor modifying the methods of using petroleum products and altering their characteristics to reduce the degree of pollution accompanying their use. Accordingly, our activities and studies have been oriented in this direction.

One of these, witn which you may be familiar, is in the development of a catalytic converter for the control of combustible contaminants from the exhaust of gasoline-burning automobiles. We were successful in developing a workable device of this type. Although it is not being accepted by the automobile industry at this time, we believe it will be important in the future. The growing severity of the automobile exhaust pollution problem will, in our opinion, soon exceed the capabilities of engine modifications to do the job alone.

This work showed that lead is the principal cause of deactivation of afterburner catalysts. We also noted the increasing frequency of questions as to the desirability of introducing lead into the atmosphere through automotive exhaust gases. Therefore, it was considered desirable that we undertake a study of the applicability of available process technology and the economic consequences which would result if the refining industry found it desirable to produce and supply the motoring public with unleaded gasoline meeting other current quality standards. The data which we are presenting today result from this study.

Our only purpose in making these data available to this Committee is to provide it with the facts and figures which we have developed in this connection. These deal with the technical feasibility of producing an unleaded gasoline and the magnitude of the refining industry's higher investment and operating costs and crude oil requirements, which would probably be reflected in the form of increased prices for gasoline sold to the motoring public.

Any evaluation of the cost to the public of eliminating lead from gasoline should, in addition to the above factors, make allowance for any changes resulting from differences in automotive performance and mileage, altered maintenance requirements, and changes in the level of emissions other than lead. However, figures on these items which are presently available to us are too preliminary to ascribe quantitative economic values to them. We will, therefore, not include them in our discussion.

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REFINING TO PRODUCE GASOLINES OF REDUCED LEAD CONTENT

M. J. Sterba,
Assistant to the Senior Vice President,
Universal Oil Products Company,
Des Plaines, Illinois

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INTRODUCTION

The anti-knock quality of unleaded gasolines is related to their chemical structure, and the intent of refining processes is in part to create gasoline by decomposing or cracking larger molecules, to direct this decomposition so that the gasoline hydrocarbons have a favorable structure, to modify the structure of already existing gasoline hydrocarbons, and to synthesize gasoline hydrocarbons of favorable structure from smaller molecules.

Paraffins, olefins, naphthenes, and aromatics are the four main hydrocarbon types of which gasolines are composed. Normal paraffins have the lowest octane numbers of the family, while the isomerized or branched paraffins have higher octane ratings. It is well known that the octane number scale has been defined by ascribing a zero rating to n-heptane which is particularly prone to knocking and a rating of 100 to iso-octane (2,2,4-trimethyl pentane), one of the more highly branched octanes. Generally, mono-olefins will have higher octane numbers than the corresponding paraffins. Naphthenes, or cycloparaffins are less prone to knock than the corresponding straight-chain hydrocarbons, and the aromatics have exceptionally high octane numbers which are over 100. Although the relationship between hydrocarbon structure and knock rating is highly involved, these broad generalizations will indicate the result that the several petroleum refining processes intend to accomplish.

The native hydrocarbon types vary widely in the proportions in which they are found in petroleum from different fields, and therefore the octane rating of the straight run gasoline will vary. However, most native gasolines obtainable by simple distillation from crude oil contain only paraffins, naphthenes and aromatics, and will have octane ratings of 50 or less. Thermal cracking which served as the main refining tool until the last World War crated gasolines that contained olefins, as well as the other three hydrocarbon types. These thermal gasolines had clear research octane ratings of the order of 75. In addition, thermal reforming was applied to the straight run gasolines to raise their octane ratings to levels as high or higher than of cracked gasolines by reactions that produced olefins from the open chain and cyclic paraffins, and that concentrated aromatics by cracking the non-aromatics into lighter fragments. These thermal processes raised the pool gasoline clear research octane number to about 75, and their application was gradually diminished during the war years.

The light olefins, propylene and butylenes, which are by-products from thermal cracking, can be polymerized under controlled conditions to form larger olefins that fit into the gasoline boiling range. These polymer gasolines have clear research octane ratings of about 95 and were helpful in raising both the quantity and quality of the refiner's gasoline pool.

Catalytic cracking, which was introduced during the last war, creates gasolines from larger molecules by catalytically controlled reactions. The paraffinic content of these gasolines is highly isomerized, and their ring content is high in terms of aromatics, cyclo-paraffins, and cyclo-olefins. Their clear research ratings are in the early nineties, and the increased application of this process during and since the war has provided a considerable boost to the octane number of the refiner's gasoline pool. In addition, the process produces propylene and butylenes which are raw materials for the synthesis of high octane gasolines.

In the years just following the war, the modern catalytic reforming processes, of which Platforming was the first and is still the most prominent process, were developed. The objective of these processes is to reform, or re-structure, the hydrocarbon types in straight run and other gasolines. The main reactions create aromatics by the dehydrogenation of cycloparaffins, and by the dehydrocyclization of paraffins having six or more carbon atoms in proper arrangement. The process therefore produces hydrogen which is useful in other refining processes. Furthermore, native aromatics are concentrated by the hydrocracking of paraffins into smaller fragments. The octane number of the sulfur-free product, or reformate, is determined by the extent to which the above reactions are carried. Clear research ratings of over 100 are practically attainable, although current operations yield reformates whose research ratings are generally in the early nineties. In all reforming processes, the yield of the gasoline product diminishes as its octane number is raised, due to the formation of light fragments by hydrocracking reactions and to the increase in density of the product. A refiner can adjust the clear octane number of his gasoline blends by the severity at which the catalytic reformers are operated.

Another catalytic process brought into operation during the war was that of alkylation. This involves the combination of iso-butane with butylenes and propylene to form an alkylate which is entirely iso-paraffinic in composition and which falls into the gasoline boiling range. Because of the iso-butane involved in the reaction, the yield of alkylate from a given volume of olefin is about twice as great as the yield of polymer when using the polymerization process. Aklylate octane numbers are of the order of 95 on a clear basis. A catalytic cracking unit does not produce enough iso-butane to react with the propylene and butylenes it also produces, but most refiners have supplementary iso-butane available from their reforming operations, or they can produce it by the isomerization of n-butane with available processes.

Processes are available and in operation today for the isomerization of paraffins heavier than n-butane. n-Pentane can be converted to iso-pentane quantitatively, thereby raising its clear octane number from 62 to 92. Similarly a 60 octane number straight run C_6 fraction can be processed to a mixed isomeric composition having a clear octane number of 80.

Hydrocracking is a catalytic process introduced within the past few years to convert distillate fractions with the aid of hydrogen to produce high yields of gasolines whose ring contents are high, although they generally require octane number up-grading by catalytic reforming. The process produces iso-butane which is useful in alkylation processes.

These are the processes which are in use or available today for the production of high octane number gasolines. Among them, the three main work horses of the refining industry are catalytic cracking, catalytic reforming, and alkylation. Eighty-five percent of our crude is processed in gasoline producing refineries having these three processes, and ninety-five percent of it is handled in refineries having the combination of at least catalytic cracking and reforming.

REFINING COST OF MAKING UNLEADED GASOLINE

Programs might be instituted whereby the lead content of gasolines could be reduced gradually over a period of time, while maintaining their octane ratings at whatever levels might be required to satisfy the existing car population. Other proposals could involve the reduction in compression ratios of manufactured engines so that they would be satisfied by the knock ratings of gasolines whose lead contents are being reduced. These approaches employ a stepwise procedure to be applied during a transition period of gradual lead reduction.

We have chosen, however, to examine the ultimate and most drastic situation where the refiner might find himself faced with the problem of manufacturing an unleaded gasoline having knock ratings of the currently marketed leaded gasolines, even though gradual transition programs might be more likely. This engineering examination was directed at a determination of how the refiner might modify his processing technique, and of what it would cost him to produce the unleaded gasoline.

Two refining situations were investigated. One study involved a U.S. average for which premium sales amount to 30 percent of the total, and another study was specific to the California region where premium sales are more than half the total. In either case, the sequence of steps employed in making the evaluation was as follows:

(a) Determine the characteristics and relative amounts of the premium and regular gasoline pools for the area under study.
(b) Establish the properties of an average crude processed in the area.
(c) Devise a simplified refining flow chart containing typical processing equipment which is operated so that the refinery produces premium and regular gasolines having properties and a quantity ratio established in (a) above. In addition, the processing is coordinated so that the yields of gasoline, distillates, and residual fuel, based on crude, represent the average refinery output for the area in consideration. This, then, is intended to represent the current refining situation, or base case, when making leaded gasolines.
(d) As a next step the refining scheme established in (c) is modified by the addition of processing equipment, and so operated in coordination with the existing equipment that unleaded premium and regular gasolines can be produced in the same proportions and having the same anti-knock properties as in the base case. Two variations were explored.
(e) By comparing the equipment and operating requirements in the processing schemes established for clear and leaded gasolines, an incremental manufacturing cost was computed for the clear gasoline, giving consideration to the involved effects of capital expenditures, operating costs, and product values.

THE U.S. AVERAGE SITUATION

So that the effect of refinery size on manufacturing costs might be estimated, refineries of 50,000 and a 25,000 barrels per stream day of crude processing capacity were considered. These capacities were selected rather arbitrarily and are not intended to represent an average or typically sized domestic refinery. It is interesting that the above capacities fall within the 20,000 to 65,000 barrel per day range in which over 30 percent of our crude is refined, and in which over 40 percent of the number of our gasoline producing refineres are found.

The U.S. Average Gasoline

Using data compiled by the U.S. Bureau of Mines, the average of the summer and winter grades of premium and regular gasolines showed research octane numbers of 99.5 and 92.5, and lead contents of 2.56 and 1.9 grams per gallon respectively at the time the study was made. (Table I.)

The Average Crude

From inspections of crudes provided by the U.S. Bureau of Mines, and considering production rates in those areas making major contributions to our crude supply, a statistically weighted average crude oil was defined. This average crude is not represented by any specific crude, but has the following calculated properties: (See also Table II.)

API	35.2
Sulfur, Wt. percent	0.63

	Vol. percent
Light Ends	3.0
Gasoline	26.4
Light Distillate	23.8
Heavy Gas Oil	34.8
Pitch	12.0

These properties resemble those of many Mid-Continent crudes.

Refining Scheme for Base Case

The sequence of refining steps, selected and organized to convert 50,000 barrels per stream day (BSD) of the above described crude into major products of current yields and properties is outlined as follows: (See also Figure I.)

1. Atmospheric and vacuum distillation resolves the crude into the nominal cuts indicated above for the crude break-up.
2. The 6000 BSD pitch is processed in a thermal cracking unit, simply to accomplish viscosity breaking. Further viscosity reduction is achieved by cutting back with clarified slurry oil and heavy cycle oil from the catalytic cracking unit to produce a No. 6 fuel oil. The small amount of gasoline produced is blended into the regular pool without further processing other than treating.
3. All of the gas oil (17,400 BSD), heavier than the 625° F. end point middle distillate is processed in a catalytic cracking unit. A conversion of 63 percent was selected to balance the proportions of overall refinery products, and represents average U.S. performance of catalytic cracking units. The debutanized gasoline is routed to both premium and regular gasoline pools.
4. All but 1000 BSD of the dehexanized straight run naphtha are Platformed to a clear research octane number of 90, and the Platformate routed to both the premium and regular pools. The combination of reforming severity and naphtha by-pass was chosen to obtain the objective octane numbers of the two gasoline pools.
5. Both the 11,900 BSD of virgin distillate and the 4002 BSD of catalytic light cycle oil were arbitrarily Unifined for sulfur removal, and combined to form a net production of distillate oils.
6. All C_4 and lighter streams are collected in a gas concentration unit which provides C_3 stream to a polymerization unit, a C_4 stream for alkylation of the butylenes to the extent of iso-butane availability, and a fuel gas stream. The 571 BSD of C_3 Polymer and the 1980 BSD of alkylate are routed to the premium pool. The 1141 BSD of propane liquefied petroleum gas (LPG) stream is made as a by-product from the polymerization unit.

By appropriately blending the several available gasoline components, the premium and regular gasoline pools in the desired ratio of 30/70 were obtained, and their linearly blended research octane numbers, vapor pressures, and lead contents were made to match those indicated earlier for the national average. The numerical details of the composition and properties of these blends is shown in Table III, which indicates that a good match with the national average was obtained. Nine-hundred-and-six (906) BSD of outside butanes were required to bring the gasoline pools to the objective vapor pressure of 10.2

The capacities of the major processing units, that is the Platformer, catalytic cracking unit and alkylation unit, expressed as a percent on crude, are in good agreement with national averages for these processes. To that extent the base case refining scheme resembles actual refining practice on an average basis. This scheme, however, was intentionally devised to produce gasoline, distillate, and residual fuel as the three net products, and therefore does not represent any specific refinery, nor is it typical of an average refinery. This was done to avoid the complication of involvement with a variety of subsidiary manufacturing steps to make a wide range of diversified products. The output from the base case matches rather well the national average refinery output on a consolidated basis, where jet fuels, kerosene, distillate fuel, lubricating oils and waxes are combined and shown as a single yield; similarly, asphalt, coke, and road oil can be combined with residual fuel and shown as a single yield. The numerical comparison is shown below, indicating good agreement of the base case with the consolidated national average refinery output derived from the latest available edition of the API Petroleum Facts and Figures.

REFINERY OUTPUT OF BASE CASE COMPARED WITH NATIONAL AVERAGE

Product as Vol. Percent of Crude	Base Case	National Average
LPG	2.2	2.4
Gasoline	49.3	48.3
Distillates	31.4	32.2
Residual Fuels	15.4	15.1
	98.3	98.0

Production of Unleaded Gasoline (Case I)

In order to produce a premium gasoline having a clear research octane rating of 99.5, there must be available a stream having an octane number of over 100 so that other streams having octane numbers less than the objective 99.5 can be blended off. Appropriate major components for this premium blend would be alkylate and a Platformate of such amount and octane number that the objective knock rating is obtained after the blend is fortified with C_5 and C_4 for front end volatility and vapor pressure. Because of its high ring content, an appropriate feedstock for the premium Platformer is the catalytically cracked naphtha.

It is noted that the light straight run C_5 and C_6 fractions are present in such quantity and are of such low octane number that it becomes feasible to isomerize these fractions for octane improvement.

The approach chosen to prepare the unleaded premium and regular pools from 50,000 BSD of crude for this Case I was to modify the base case flow diagram, or to insert new processing equipment as follows:

1. Install a Penex unit to isomerize the straight run C_5 cut in recycle operation to isopentane, and the C_6 cut in once-through operation to improve the octane rating of both cuts.
2. Install a new Unifiner and Platformer to process a depentanized proportionate blend of catalytically cracked and thermal gasolines. By successive approximation it was determined that the required operating severity was to produce a Platformate having a clear research octane number of 102.
3. A depentanizer is provided for the cracked gasolines to furnish a high octane number light end blending component.
4. A distillation column is installed to re-run the high octane Platformate made in Step 2 above, in order to be certain that high boiling fractions are not present in the premium gasoline.
5. The existing Platformer in the base case flow diagram is modified to produce a 94 clear octane number from all (10,650 BSD) of the straight run naphtha.

The remainder of the processing units in the base care are left undisturbed with respect to capacity and operating severity. For this Case I the basis of clear gasoline manufacture was to produce the same quantities of residual fuel and distillates as were made in the base case, but to allow the volume of total gasoline to decrease due to the increase in Platforming severity required to raise the overall gasoline pool research clear octane number from 88.4 to 95.3 in moving from the leaded to this clear case. The volume of the clear gasoline pool is about 90 percent of what it was in the leaded case, but the proportions of premium and regular gasolines are unchanged.

With the Platformer feeds and severities set as indicated above, the various other gasoline streams are combined with these Platformates of 102 and 94 clear octane numbers to form the unleaded premium and regular grade pools as shown in Table IV. The octane numbers of these unleaded premium and regular blends match those of the national average gasolines, while their sulfur contents are distinctly lower. No outside C_4 is required to bring the gasoline pools to the objective vapor pressure of 10.2.

As pointed out above, the total clear gasoline pool in this case is about ten percent smaller in volume than it was when leaded gasoline was produced. It is of interest to examine the economics of a refining scheme for bringing the volume of gasoline back up to what it was in the base case, but when still processing only 50,000 BSD of crude oil. This study is the basis of the next case, and assumes that the crude units and the cracking units are operating at full capacity, so that they can process no more feedstock, nor can the catalytic cracking unit be operated at a higher conversion.

Production of Unleaded Gasoline (Case II)

The approach used in this study was to hydrocrack (Isomax) such an amount of the straight run distillate that after the hydrocracked gasoline is Platformed to the proper octane level, the volume and proportion of the premium and regular gasolines are restored to what they were in the base case of leaded gasoline manufacture. Two-thousand, three-hundred forty-nine (2349) BSD of additional clear gasoline must be produced, above what was made in Case I. This is accomplished by inserting an Isomax unit into the process flow scheme of Case I to process 2616 BSD of straight run distillate. The hydrocracked

C_5 and C_6 fractions are routed directly to the regular gasoline pool, while the dehexanized hydrocracked naphtha is reformed in about equal amounts in two Platformers producing 102 and 94.6 octane number gasolines for the premium and regular pools respectively.

The remainder of the refining scheme is essentially the same as for Case I, with the exception that the distillate product has been diminished by the amount of the Isomax unit feed. The Platforming units produce sufficient hydrogen for the hydrocracking and for the rather extensive Unifining of the entire distillate pool. Because of additional iso-butane generated by the Isomax unit, some of the propylene was diverted from polymerization to alkylation so that additional barrels of high quality gasoline could be made.

Numerical details of the composition and properties of the blended premium and leaded gasolines for Case II are presented in Table V, and in which it is noted that the octane numbers of these blends match those of the national average, but that their sulfur contents are much lower.

Summary of Processing Studies

For the three cases studied, the overall refinery input and product yields are brought together in Table VI, representing the situation before steam generation or the thermal requirements of the processing units are met. All of the fuel gas and some of the heavy fuel oil must be burned to meet these requirements.

No attempt has been made to optimize the two approaches for making unleaded gasoline from the standpoint of finding the most economical combination of feed rates and product octane numbers for the two Platformers. Furthermore, other refining schemes could have been investigated. For instance, an alternate to Case II might have involved further conversion of the pitch to bring the volume of gasoline production back to what it was in the base case, instead of restoring the gasoline production by hydrocracking the distillate. This could have been accomplished by delayed coking of the pitch to produce additional gas oil for catalytic cracking. Another approach would have been to employ catalytic processes that are now becoming available to hydrocrack the pitch to accomplish desulfurization, viscosity reduction, and gasoline production simultaneously. This gasoline would require further processing as it did in Case II. The best approach to the modification of a refining flow diagram to produce unleaded gasoline will depend on the particular existing combination of processing equipment and the schedule of products made. It is of interest that about 85 percent of the crude is processed in refineries having the particular combination of catalytic cracking, catalytic reforming, and alkylation; and 95 percent is handled in refineries having catalytic cracking and reforming. The general principle of reforming the cracked gasolines will be applicable, then, in the majority of the specific refining cases to be considered.

Platforming operations at the indicated severity of 102 clear octane number are not far removed from certain commercial experiences. We have data on record from a Platformer which has been in sustained operation over a period of several years producing a Platformate having a clear octane number of 99 from a straight run naphtha having a less favorable ring content than the cracked naphtha proposed in this study for the 102 clear octane number operation. Other units have experienced extended runs while making Platformates of 97 to 98 clear octane number. Therefore, with commercial experience at severities close enough to the projected 102 octane number, it is considered to be a practical and feasible commercial operation. Furthermore, catalytically cracked naphthas have characteristically high ring contents, and are therefore easier to reform than are the straight run naphthas with which the refining industry is most familiar.

In matching the research octane number of the premium and regular pools with those for the national averages, linear volumetric blending of the octane numbers of the components was employed. However the laboratory octane numbers shown for the premium and regular gasoline blends for the base case and the two unleaded cases in Tables III, IV, and V were computed by using comprehensive blending programs which take into account the chemical composition of the components. These octane numbers are higher on a research basis and lower on a motor basis than those obtained by simple linear volumetric calculation. The more important comparison should be made on the basis of road octane ratings of the clear blends and the national average. Regardless of which of the several methods are used for estimating the road ratings from the laboratory ratings, there is a very close correspondence between the clear gasoline blends and the national average on the road rating basis.

REFINERY ECONOMICS

This phase of the study involves the computation of the manufacturing cost of the total unleaded gasoline pools of Cases I and II, as an additional incremental cost above the base case making the leaded gasoline. Table VII is a summary of these incremental cost calculations, brought up to current price situations.

Investments

The items included in the incremental investment costs are the figures for the new process units to be added, the modification of the straight run naphtha Platformer, and for offsite facilities, all on an erected basis. Provision is made in the offsite figures for additional cooling water and boiler facilities, intermediate tankage, yard piping and paving. The figure for catalysts represents the value of the initial load of catalysts for the new processing units and includes the platinum value at current prices of the Platforming catalyst. Working capital represents the value of charging stock and refined products accumulated over a 20-day period in order to put the refinery on an equilibrium inventory operating basis. On an incremental basis the working capital number is negative because of smaller product volumes in Cases I and II.

Operating Costs

The unit values used for the cooling water and steam represent only direct operating costs to produce these utilities since the indirect costs are taken into account by showing depreciation and interest charges on offsites as a separate item. The unit value used for the fuel is equivalent to $2.38 per barrel of No. 6 fuel oil, and the amount represents its use for process heating requirements, but not for steam generation. Royalties are figured on a running basis for Penex, Platforming, Isomax, and Unifining, all UOP units. Estimated operating labor requirements are four and seven additional men per shift for Cases I and II respectively, and one extra supervisor for either case. The general overhead figure includes items related to additional office help, gaging, yield accounting, and process engineering. Straight line depreciation was used over a 10-year period on the process unit investment, and over a 20-year period on offsite investment. However, a five percent annual interest charge is shown on the entire investment. Maintenance, local taxes, and insurance are figured at a combined annual rate of seven and one-half percent on the process and offsite investment.

Summary of Manufacturing Costs

The concluding part of Table VII is a summary of values represented by the feed, refined products, and operating costs, all on an incremental basis. Quantities of charge and products for the three cases discussed above are shown in Table VI, and their unit prices were selected as representing a weighted average on a national basis. Crude prices are not involved since the amount is constant for the three cases considered. The prices used for the computation were as follows:

Premium Gasoline,	¢/Gal.	13.4
Regular Gasoline,	"	11.6
Distillate,	"	9.6
Residual Fuel,	$/Bbl.	2.38
Butanes - 65 percent normal,	¢/Gal.	6.5
- 90 percent normal,	"	5.25
LPG,	"	4.0

A negative value of $2470 per day shown for the charge stock is the cost of the butanes that were purchased in the base case for vapor pressure, but are not required in the two cases of unleaded gasoline manufacture. The refinery product values are shown to be negative since there is a smaller volume of either gasoline or distillate produced when making unleaded gasoline. Arithmetically, the term "net incremental manufacturing cost" as shown is the difference between the operating cost plus product value and the charge stock value. This, then, represents the additional cost of manufacturing the unleaded gasoline if it were sold at current prices. If, however, the unleaded gasoline was priced at 1.13 and 1.1 cents per gallon more than current prices for Cases I and II respectively, these incremental manufacturing costs would reduce to zero.

A separate study was made for a particular refiner whose premium gasoline output was about 90 percent of the total sales, using the process flow arrangement, crude, and price structure specific to this particular refiner. In this unusual instance the estimated incremental costs of making unleaded gasoline were double those of the corresponding U.S. average cases, and the study points out the strong influence of the octane level of the overall gasoline pool on these costs.

Smaller Refinery Economics

Unleaded gasoline manufacturing costs were computed for a 25,000 BSD refinery having exactly the same processing arrangement as the larger 50,000 BSD refinery just studied. Using the same crude and yield structures for the half-size refinery, the cost items shown in Table VII were re-calculated. For the smaller refinery the added investment costs amount to $5,710,000 and $8,091,000 for Cases I and II respectively. Labor, supervision, overhead, and laboratory charges were the same as for the larger refinery. Using the same rates for depreciation and interest, and the same product unit prices as for the larger refinery, the incremental manufacturing costs amount to 1.41 and 1.50 cents per gallon of gasoline for Cases I and II when processing only 25,000 BSD of crude.

REFINERY ECONOMICS IN CALIFORNIA

It is of interest to examine the economics of unleaded gasoline manufacture in the California region where the premium grade represents over one-half of the total gasoline sales, contrasted with about 30 percent premium sales on a national average. For this study a percentage of 55 percent premium was used, and the procedure as applied to the processing of 50,000 BSD of crude was the same as for the national average study. Similarly the refining processing arrangement was the same as for corresponding cases in the national average.

The crude oil used for the California study was heavier than the national average, having an API gravity of 28, a vacuum pitch content of 22 percent, and a sulfur content of 1 percent.

In all cases the catalytic cracking conversion was set at 67 percent, and for the base case the Platforming unit was required to produce a 94 clear octane number gasoline in order to meet the pool anti-knock requirements.

For the cases of making unleaded gasoline, the Platformer processing the cracked gasolines was operated to make a 102 research octane number gasoline, while the unit processing the straight run naphtha produced a 95 octane number Platformate.

Economic calculations for the California situation were made in the same manner and detail as for the U.S. average study. Investment costs were higher for the California cases primarily because of the required higher capacity of the Platformer processing the cracked gasolines to the 102 octane number to make the premium gasoline in a higher proportion than for the national average. Unit prices used for the refined products in the California area were about the same as for the national average, except that the distillate fuel price was 8.1¢/Gal. instead of the 9.6¢/Gal. figure used for the national average.

The additional cost of manufacturing unleaded gasoline in the California study amounts to 1.36 and 1.11¢/Gal. of gasoline for Cases I and II respectively. Thus, the conversion of some of the distillate into gasoline in Case II for the California study is relatively more attractive to Case I than it was in the national average study where the price differential between gasoline and distillate was smaller.

SUMMARY

In the variations of the generalized situations considered in this discussion, the estimated incremental manufacturing cost to the refiner for making an unleaded gasoline of current octane number quality ranged from 1.1 to 1.5¢/Gal. above what it costs to make current gasolines. This incremental cost will vary, both above and below the range indicated, depending on the refinery size, its type, its location, crude characteristics, crude supply situation, products made, price structures, labor costs, accounting methods, and many other factors.

The important conclusion to be made is that it is possible to make unleaded gasolines, but at costs that must be passed on to the driving public.

In order to market the same amount and quality of clear gasoline as the refiner presently sells of leaded gasoline, he must either market less of distillates or residual fuel, or process more crude oil.

To market the same amounts of clear and leaded gasolines and, in addition, the same amounts of total liquid products in the two situations, an increase in crude throughput of about two percent is indicated by these studies for the leaded situation.

Based on estimates made for a 50,000 BSD refinery, the U.S. refining industry would be required to make a capital investment of 1.5 to 2 billion dollars. This is to be compared with annual expenditures of the order of 500 million dollars for normal refinery expansion and modernization.

Unleaded gasolines of the compositions discussed have been prepared, and in driving tests covering tens of thousands of miles their general performance was equal to and in some respects better for the clear than for the corresponding leaded gasolines.

TABLE I

Properties of the U.S. Average Gasoline

	Premium	Regular
API	61.4	61.9
Sulfur, Wt. percent	0.03	0.052
Lead, gm./gal.	2.56	1.9
Octane Numbers		
Research	99.5	92.5
Motor	90.5	84.5
RVP	10.2	10.2
Distillation, °F.		
10 percent	120	119
30	166	160
50	214	207
70	256	261
90	323	338

TABLE II

Properties of the U.S. Average Crude

	Wt. %	API	Vol. %	% S
Loss	0.2		0.4	
C_3	0.2		0.4	
i-C_4	0.3		0.5	
n-C_4	1.2		1.7	
i-C_5	0.7		0.9	0.01
n-C_5	1.2		1.6	0.01
C_6	2.1	78.1	2.6	0.02
Gasoline, C_7-400° F	19.2	53.3	21.3	0.03
Distillate, 400-625° F	23.3	38.7	23.8	0.25
Heavy Gas Oil	37.3	24.0	34.8	0.90
Pitch (100 Pen.)	14.3	8.5	12.0	1.60
Crude	100.0	35.2	100.0	0.63

TABLE III

Composition and Properties of Gasolines Produced in Base Case

Composition, BSD		Premium	Regular
Outside Butanes	906	664	1,549
Butanes from Alky Unit	1307		
Straight Run C_5		500	750
Straight Run C_6		--	1,300
Straight Run Naphtha		--	1,000
C_3 Polymer		571	--
C_4 Alkylate		1,980	--
Thermal Gasoline		--	581
Platformate		1,908	6,120
Catalytically Cracked Gasoline		1,908	6,270
Total BSD		7,531	17,570
Volume Percent of Total		30	70
Properties			
API		62.6	58.0
Sulfur, Wt. Percent		0.026	0.051
TEL, ml./Gal.		2.42	1.8
Octane Number, Leaded			
Research		99.5	93.9
Motor		91.2	83.9
Octane Number, Clear			
Research		92.8	86.6
Motor		84.0	77.4
RVP		10.2	10.2

TABLE IV

Composition and Properties of Unleaded Gasolines Produced in Case I

Composition, BSD	Premium	Regular
Butanes from Alky Unit	634	1,055
C_5/C_6 from PENEX Unit	--	2,585
Cat. Cracked C_5	503	743
C_3 Polymer	--	540
C_4 Alkylate	1,223	757
Thermal Gasoline	--	116
Catalytically Cracked Gasoline	--	1,636
Platformate (102 Octane)	--	8,490
Platformate (94 Octane)	4,470	
Total BSD	6,830	15,922
Volume Percent of Total	30	70
Properties		
API	53.5	59.6
Sulfur, Wt. Percent	0.0017	0.014
TEL, ml./Gal.	0	0
Octane Number		
Research	100.1	93.3
Motor	89.8	84.3
RVP	10.2	10.2
Distillation, °F.		
10 Percent	114	117
30	178	142
50	217	215
70	253	270
90	329	336

TABLE V

Composition and Properties of Gasolines Produced in Case II

Composition, BSD	Premium	Regular
Butanes from Alky Unit	693	1,107
C_5/C_6 from PENEX Unit	--	2,585
Isomax C_5/C_6	--	777
Cat. Cracked C_5	555	691
C_3 Polymer	--	417
C_4 Alkylate	1,248	1,032
Thermal Gasoline	--	116
Catalytically Cracked Gasoline	--	1,636
Platformate (102 Octane)	5,035	
Platformate (94.6 Octane)	--	9,209
Total BSD	7,531	17,570
Volume Percent of Total	30	70
Properties		
API	53.0	59.4
Sulfur, Wt. Percent	0.0017	0.013
TEL, ml./Gal.	0	0
Octane Number		
Research	100.1	93.3
Motor	89.8	84.0
RVP	10.2	10.2
Distillation, °F.		
10 Percent	115	119
30	178	144
50	218	211
70	255	266
90	329	334

TABLE VI

Summary of Refinery Input and Product Yields

(U.S. Average)

	Base Case		Case I		Case II	
	BSD	Vol. %	BSD	Vol. %	BSD	Vol. %
Input						
Crude	50,000	98.2	50,000	100.0	50,000	100.0
Butanes (65 percent normal)	906	1.8				
	50,906	100.0	50,000	100.0	50,000	100.0
Output						
Fuel Gas	(19,760)*		(29,570)*		(32,640)*	
C_3 LPG	1,141	2.2	1,602	3.2	1,787	3.6
Butanes (90 percent normal)	--	--	155	0.3	282	0.6
Gasoline	25,101	49.3	22,752	45.5	25,101	50.2
Distillate	15,940	31.4	15,960	31.9	13,350	26.7
Residual Fuel	7,836	15.4	7,836	15.6	7,836	15.7
		98.3		96.5		96.8

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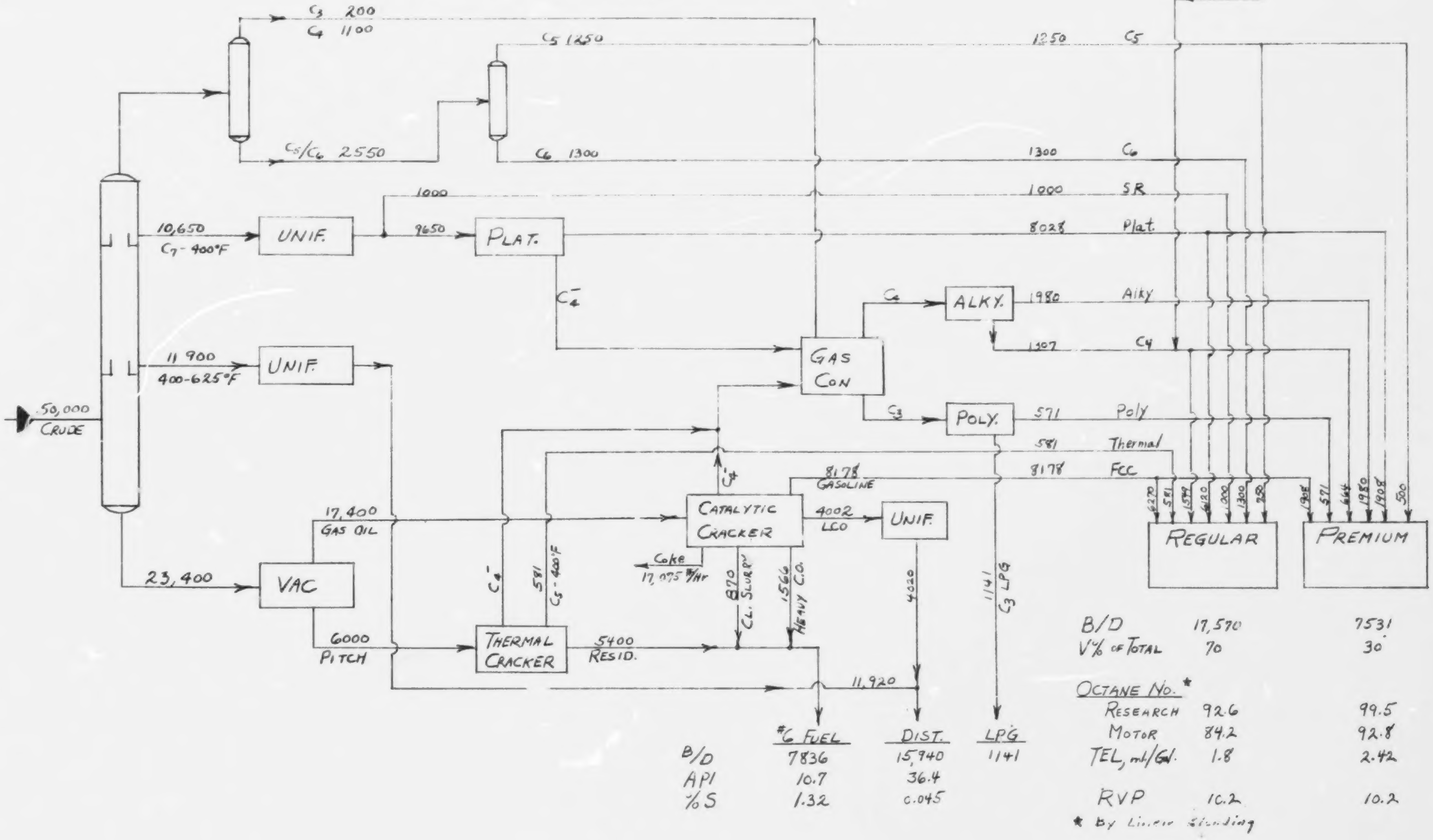
* Pounds per hour.

TABLE VII

Summary of Incremental Costs for Average U.S. Refinery of 50,000 B/SD Crude Capacity

Case	Base	I	II
Gasoline Produced, Gal./Day	1,054,200	955,600	1,054,200
Incremental Investment			
Process Units		$5,090,000	$7,150,000
Offsites		1,940,000	2,600,000
Spare Parts (3% of Process Units)		153,000	215,000
Initial Catalyst Loading		699,400	921,800
Start-up Expenses		115,000	147,000
Working Capital		(210,000)	(190,000)
Total Incremental Investment	0	$7,787,400	$10,843,800
Incremental Operating Costs, $/Day			
Fuel, 36.5¢ per 10^6 BTU		1,140	1,660
Electricity @1¢ per KWH		600	980
Cooling Water @ 1¢ per 1000 Gal. Circ.		80	110
Steam @ 50¢ per 1000 pounds		420	420
Catalyst Replacement		1,190	1,420
TEL @ 0.21¢ per ml.		(4,400)	(4,400)
Royalties (U.O.P. running)		460	700
Operating Labor @ $4.20 per Hr.		400	710
Supervision @ $5.80 per Hr.		140	140
General Overhead @ 10% of Labor & Super.		60	90
Laboratory		130	210
Maintenance, Taxes & Insurance		1,450	2,000
Depreciation		1,670	2,320
Interest, 5% on Total Incremental Inc.		1,070	1,490
Total Incremental Operating Costs	0	4,410	7,850
Incremental Values, $/Day			
Refinery Charge Stock		(2,470)	(2,470)
Refinery Products		(8,810)	(6,250)
Operating Costs		4,410	7,850
Net Incremental Manuf. Cost			
$ per day		10,750	11,630
¢ per gallon gasoline		1.13	1.1

REFINING SCHEME FOR BASE CASE (LEADED GASOLINE)
U.S. AVERAGE
FIG. I


50,000 CRUDE
10,650 C7-400°F
11 900 400-625°F
23,400
UNIF.
UNIF.
VAC
17,400 GAS OIL
6000 PITCH
C3 200
C4 1100
C5/C6 2550
C5 1250
C6 1300
1000
9650
PLAT.
C4-
THERMAL CRACKER
C4-
581 C5-400°F
5400 RESID.
CATALYTIC CRACKER
C3+
Coke 17,075 #/Hr
870 CL. SLURRY
1566 HEAVY C.O.
8178 GASOLINE
4002 LCO
UNIF.
4020
11,920
GAS CON
C4
C3
ALKY.
POLY.
1141 C3 LPG
C4 906
1250 C5
1300 C6
1000 SR
8028 Plat.
1980 Alky
1307 C4
571 Poly
581 Thermal
8178 FCC
6270
581
1549
6120
1000
1300
750
1908
571
664
1980
1908
500
REGULAR
PREMIUM
#6 FUEL
DIST.
LPG
B/D
API
%S
7836
10.7
1.32
15,940
36.4
0.045
1141
B/D 17,570 7531
V% OF TOTAL 70 30
OCTANE NO.*
RESEARCH 92.6 99.5
MOTOR 84.2 92.8
TEL, ml/Gal. 1.8 2.42
RVP 10.2 10.2
* By Linear Blending

STATEMENT

P. N. Gammelgard,
Chairman, Air-Water Conservation Committee,
American Petroleum Institute,
New York, New York

A month ago I joined the staff of the American Petroleum Institute to head its expanded activities in the fields of air and water conservation. My industrial experience for the past thirty years has been in petroleum refining. I served on the Surgeon General's National Advisory Committee on Community Air Pollution, and for the past six years on the Board of Trustees of the Northwest Community Hospital, Arlington Heights, Illinois.

The various disciplines represented by the participants in this symposium have been stimulating to me, an engineer by profession. I have not understood all that I've heard, and then maybe not all of you understood what you heard yesterday afternoon about the economics of petroleum refining with or without lead.

The widely ranging answer to what might appear to you to be a very simple question--namely, what would it cost to take the lead out of gasoline--even confused me.

I said yesterday afternoon the petroleum industry would work out its own economics; and, believe me, the task is a formidable one.

Truly there is no such thing as a typical refinery. And on this point UOP, Ethyl, and Houston Chemical agree.

Now, whether the answer comes out that the additional industry-wide investment in refining facilities for making unleaded gasoline is $1 billion or $5 billion, or whether the incremental costs, operating costs, of making lead-free gasoline is 1 cent a gallon or 4 cents a gallon, I think we are putting the horse before the cart in even bringing up such investment and cost figures in this symposium, when the conclusions and recommendations of the sessions are not yet arrived at.

In his opening remarks I recall Dr. Gehrig, the Deputy Surgeon General, saying something along the following lines. Our knowledge is far from adequate. There are important differences of opinion as to whether there are hazards to the public. Diametrically opposite viewpoints exist. And all too often industry and government do not keep up with problems created by technological developments. After listening to the discussions on Monday and Tuesday, it is apparent to me that Dr. Gehrig knew what he was talking about.

I do not believe that the widely divergent views on a number of matters were resolved. So I would therefore anticipate that in such cases there will be recommendations forthcoming for needed research.

I for one gather that more work needs to be done to reach an objective definition of the role of lead in the environment as it affects health and welfare.

I am not proposing that we research the subject to death. And I will admit that at times industry has been guilty of this very tactic. But neither do I propose that action should be taken in either reducing or eliminating lead from gasoline before we have sound data on which to base action. And, in my opinion, such data have not been presented in this symposium, nor elsewhere.

The oil industry, as I said, will develop figures for various sizes of refineries and operating under various conditions. Based on my thirty years experience in refining I will make the flat prediction that a regulation requiring the industry to make lead-free gasoline will drive dozens of the nation's 270-odd refineries out of business overnight.

Public health is without question the most vital consideration. But let us be certain that surveillance data show a trend that the margin of safety with respect to public health is actually being significantly and/or seriously narrowed before taking any steps that would result in (1) shutting down many plants, both refineries and lead plants, and depriving thousands of their jobs; (2) cause the capital drain of investment of billions of dollars in additional refining facilities and, conversely, make idle many hundreds of millions at least of facilities that would be shutdown; (3) increase the price of gasoline to the consumers. I would like to just point out that there are about 70 billion gallons of motor fuel sold in the United States per year. This is an astronomical figure. And at 2 cents a gallon--just let's pick that one out of the air--the added cost if all the 2 cents are passed on to the consumer, which we presume would be the case, would be, by simple arithmetic, $1.4 billion per year.

One word about this factor of safety. We in refining are certainly well aware of the importance of the factor of safety, and would be the last people in the world to say that there shouldn't be one in health as in engineering.

A sweeping program of research projects in the field of air conservation was unanimously approved by the Board of Directors of API early last month. Funds exceeding $2 million have been provided for the year 1966. And this is equal to our program of research in the past ten or fifteen years.

The Board indicated its willingness to provide similar sums in future years for worthwhile projects. Individual oil companies have programmed for 1966 an additional $41 million for air research and air pollution control facilities, with by far the greater part of that money going into air pollution control facilities, not just in refineries but in other divisions of the oil companies.

Our industry intends to keep up with the problems created by technological developments. We will review our program in the near future with the Public Health Service. We hope that a number of projects can be carried out cooperatively with PHS and other interested groups. The results of this research will, of course, be made available to PHS.

We have a number of research projects in the field of lead. Several of these will be placed with universities and research institutes. And I would certainly expect that our program in lead may well be influenced and reshaped by the recommendations for research which this symposium will bring forth.

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PANEL SUMMARIES AND DISCUSSIONS

Chairman: V. G. MacKenzie
Co-Chairman: A. J. Haagen-Smit

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MAJOR SOURCES OF LEAD POLLUTION

Robert J. M. Horton, M.D.,
Chief, Field Studies Branch,
Division of Air Pollution,
Public Health Service,
Cincinnati, Ohio

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1. LEAD EXPOSURE FROM AIR: Air pollution levels for lead at ordinary urban measuring stations show values ranging from 1 to 3 micrograms per cubic meter of air. Rural values are normally below these levels. Measurements taken near automobile traffic may be as high as 40 micrograms per cubic meter depending on proximity to the roadway and traffic density. High levels may also be found near some industries which employ or produce lead. On the basis of presently available data it appears that the principal source of atmospheric lead is the combustion of leaded gasoline. For example, in a representative community such as Cincinnati, 1700 pounds of lead are contributed to the air each day from this source. Combustion of coal in the same city contributes between 5 and 10 percent of this amount. Other fuels such as oil and gas do not make significant lead contributions to the atmosphere.

The contribution of specific industries to lead air pollution is poorly documented. Emission studies are needed to clarify their role. In addition, the lead emissions from rubbish combustion need further study. This applies not only to general rubbish burning at dumps and incinerators, but also to special situations such as burning of demolished houses and waste batteries and automobiles. At present these sources do not appear to contribute substantially to general urban lead air pollution. They are, as is the automobile, capable of producing intense local problems.

Some sources of lead air pollution have increased with the passage of time (for example, leaded gasoline) and others have decreased (for example, coal). It is very difficult, if not impossible, to determine with certainty whether general urban air lead levels have changed over thirty or forty years due to the lack of strictly comparable measurements. Changes occurring in recent and future years need further study. It has been suggested that city-center values may be declining with the development of suburban industrial and shopping centers.

Another area needing investigation is that of the chemical composition and particle size distribution of lead generally, and from specific emission sources.

2. LEAD EXPOSURE FROM FOOD: Available evidence indicates that lead is widely distributed in all natural foods. The average daily diet contains about .3 mg of lead per day, with a range from .1 to 2 mg. It is probable that the average lead intake from food has changed very little during the past thirty years. Possibly there has been some decline in levels recently, but this is still uncertain. The decreasing use of soldered food containers and cooking utensils, and the decrease in the use of leaded insecticides, make a lessening exposure through foods probable. In addition, the modern practice of coating the interiors of tin cans has reduced the contact of canned foodstuffs with the solder which is still used in these containers.

Geological prospecting by means of metal levels in plants has shown that lead levels in vegetation, including vegetable crops, may be high not only near lead-bearing ores but also adjacent to highways. While these lead levels are somewhat reduced by washing the plants, the remaining lead content is well above that ordinarily found.

Recent studies made on shellfish show that these animals, unlike most living forms which have been studied, selectively remove lead from water. They may achieve remarkably large body burdens of lead under certain circumstances when this metal is available to them.

These studies are not only of interest in relation to human food supplies, but also have great biologic interest in general, and information on lead in the shells may have wide application.

Since lead in milk, like lead in blood, is representative of the body's burden of lead, it was suggested that lead levels in milk might offer a useful survey tool for representing the lead exposure of cattle.

3. LEAD CONTAMINATION OF WATER: Contamination of drinking water by various sources was reviewed in terms of the Public Health Service drinking water standards of .05 mg per liter. The following items were noted:

a. The contribution of motorboats, other than for small water bodies, is not significant when compared to the contribution by sewerage.

b. Sewage treatment processes result in removal of about 15 percent of the lead for primary treatment processes, and about 50 percent for secondary treatment processes.

c. Lead is frequently present in unfiltered surface waters in quantities well above the drinking water standard. However it is noted that only a fraction of this lead is dissolved, and that low turbidity is an important property that can minimize exposure to the consumer.

d. Lead in treated surface waters is generally below the Drinking Water Standard. Only 5 of 876 samples analyzed by the Water Pollution Surveillance System of the Public Health Service in 1962 exceeded .05 mg per liter. Streams in many localities where opportunities for enrichment in lead content might be expected to occur did not show appreciable quantities of lead.

e. The lead content in municipal water used for interstate carrier drinking water supplies indicates little need for concern about the lead content. Only 4 of 531 samples showed a lead content in the .03 to .039 mg per liter range.

f. Lead in rain water collected directly from the atmosphere varied in lead content up to 0.49 mg per liter, depending on the nearness to highways and other sources of contamination, and in general was highest where maximum opportunity existed for the inclusion of atmospheric dust.

g. The use of lead compounds in direct contact with drinking water indicated several sources: lead chromate paints, lead pipe for service use, lead containing solders for copper distribution systems.

Appropriate questions for consideration in future studies relative to lead contamination of water were summarized as follows:

a. Pick-up of lead from solder and from soldered copper tubing distribution systems.

b. Exposure of householders to lead from water in older housing which still retains lead piping.

c. Exposure of householders to lead from cistern supplies.

d. Intensified surveillance of interstate carrier water supplies, surface water supplies, and precipitation.

4. OCCUPATION EXPOSURE TO LEAD: A review was made of mostly hitherto unassembled and unpublished data on lead exposure as measured by concentrations of lead in air and urine in seven or eight work categories representing the major uses of lead: auto body manufacture and repair, foundries, electrotype printing, storage battery manufacture, rebuilding of storage batteries, and secondary smelters. The large category of munitions was not included.

Insofar as the concentration of lead in air and urine can be used as measures of exposure to inorganic forms of lead, the clear, consistent, and unmistakable conclusion is that exposure to airborne lead dust and fume is less by a factor of several magnitudes in the industrial categories since 1934. This decrease has continued, but at a slower rate, since 1945, at which time the average exposure in most work categories was below the acceptable limits owing to the introduction of safe work standards for lead in the air and in urine.

Of a ninth work category involving exposure to gasoline additives and their breakdown products, organic and inorganic lead, which includes gas and service station attendants, refinery handlers, garage mechanics, parking attendants, traffic officers, and motor vehicle drivers, only one--garage mechanics--showed evidence of exposure (elevated urinary lead values) slightly in excess of normal, but still well below acceptable limits.

It was noted that although the trend has been toward overall improvement in occupational exposures where attention has been paid to the problem, there are still many small, and a few large, industrial establishments which are in need of improved occupational health practices. It was noted further that the number of workers exposed to high levels of lead had probably not decreased significantly in the past twenty years due to the increase in small, poorly controlled establishments, although most of the larger establishments had instituted satisfactory occupational hygiene practices.

The need was expressed for improved and standardized reporting practices to allow comparison of occupational health statistics nationwide. Data is poor or lacking on the proportion of the 70 million employed persons in the United States who are exposed to lead, and on the trends in many types of exposures which may be occurring.

5. LEAD EXPOSURE FROM OTHER SOURCES: Cigarettes represent a minor but common source of lead. While most of the lead in tobacco is known to remain in the ash, a small proportion is found in the smoke. This is sufficient to offer a lead intake from this source probably of about 20 micrograms per pack. The retention rate is probably quite high since the particle size of tobacco smoke is small.

During the economic depression of the 1930's there were a number of outbreaks of severe lead poisoning which resulted from burning of discarded battery cases in household stoves. Such episodes have become less frequent in homes and are now rare.

However, modern battery cases are sometimes disposed of by burning in dumps and may be a source of community exposure to high air lead levels.

An important, though diminishing source of lead exposure is that of lead paints in pre-school children. At this age the child who is not carefully tended will commonly eat paint by putting peeled flakes into his mouth or by gnawing on painted surfaces. Lead-containing paints are no longer used on toys or homes. However, old houses commonly still contain such paint and afford opportunities for poisoning. In older cities this is the most frequent fatal poisoning found in children. Many of these cases are discovered too late in their course for effective treatment. Improvement of early case finding methods is urgently needed.

The use of pb 210 and other isotopes was discussed briefly in relation to body content and also as tracers in biological studies.

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DISCUSSION

MR. CHOLAK: A question dealing with the problem of the amount of lead retained by cigarette smokers: I think that most of the calculated data takes into account the total amount of lead in tobacco. We have found that about 5 percent, maybe a little less, of the total lead in tobacco enters the smoke. On analysis of a number of samples, the average concentration of about 1.6 micrograms of the total lead in a cigarette was found to be present in tobacco smoke.

I haven't ordinarily made it a point to observe how smokers handle their cigarettes. I watched one fellow on the plane for a complete minute. He took two puffs on his cigarette, each puff lasting no more than two or three seconds. On the basis of that calculation I figured that the amount of lead that this individual inhaled was no more than 2 micrograms. Probably, on the basis of the retention of smoke in the lungs he didn't retain more than 0.6-0.7μ of lead.

DR. KEHOE: There was one point in particular in Dr. Horton's report that I want to call attention to, because I think, if I understood what he said, it should be modified. I'm very sensitive on the point of the amount of lead that is natural in food, having been recently somewhat sensitized to this point after a good many years of being rather careful in pointing out that much of this is the result of contamination in a variety of ways.

With respect to the quantities found in food, Dr. Horton used the word "natural," I believe. This he has got to look out for. He'd better say "total lead" or perhaps use the conventional form of "natural" or "normal" in quotes.

DR. RADFORD: I would like to make one comment on what Mr. Cholak said. It bears on the matter of retention of particulates by the body as distinct from deposition of particulates in the lungs.

First, with regard to cigarette smoking, approximately one-half of the total smoke appears in the main stream despite the fact that the individual takes about one puff a minute. There has not been any fractionation of the lead between sidestream and mainstream smoke. I don't know that we can put absolute figures on this.

However we can minimize the cigarette as a source of lead. I would say that, from our studies of polonium as a tracer, it is quite likely that a very high fraction of any of the metals that might get into the smoke, if they are in an insoluble form, would be cleared by way of the G-I tract. I think whenever we are talking about insoluble lead forms, as distinct from soluble ones, we would have to establish precisely the fraction retained within the lung tissues, or within the whole body, as distinct from the fractions that are deposited.

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RISK OF EXPOSURE AND ABSORPTION OF LEAD

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The first presentation of the afternoon was made by Dr. Tomashefski of Ohio State University. It was in reference to the inhalation of lead and the hazards involved. After some brief comments about the anatomy of the lung and the physiology of respiration, he made the following points:

First he talked about the major sources of lead in the air--this has already been covered this morning by Dr. Horton in his Summary Report, and I shan't repeat these. Dr. Tomashefski then went on to discuss particulate lead in the air.

He pointed out that whether any particulate reached the lungs depended, in the main, upon the mean size of the particles. Larger inhaled particles remained on the walls of the upper airways, and from there were carried to the pharynx by ciliary action, cough, and peristalsys. Most of this was then swallowed or spit out.

Smaller particulate lead compounds that reached the alveoli were absorbed directly from there into the circulation.

He remarked that about 75 percent of particulate lead from automobile gasoline combustion was less than 0.90 micra in mean diameter, a size that easily reaches the alveoli.

Dr. Tomashefski indicated there was no direct action of the lead compounds in the air on the lung itself. He specifically noted that there was no evidence that lung cancer was due to inhaled lead compounds.

At this point Mr. Elmer Robinson, of Stanford Research Institute, Palo Alto, presented some data on the size distribution of some 70 different samples of lead aerosols which were collected in at least eight different communities at the breathing level or, as he said, the eye level.

He noted that 75 percent by weight of the particles had a mass median diameter of less than 0.42 micra, a size that would easily reach the alveoli.

He indicated that the place of collection made no difference in the size distribution of the particles that he found. And these places ranged from rural areas to busy urban express highways.

At this point, in connection with a question that arose about body response to stored lead, Dr. Robert Kehoe, University of Cincinnati, remarked that lead metabolism was only slightly modified, if at all, by relatively severe alterations in body functions. As an example, he noted that an acute alcoholic bout in an individual does not cause any significant abnormality in his handling of his stored lead. He remarked that he knew of only two situations that caused significant changes in lead metabolism. These were the use of chelating agents and when severe dissolution of the human skeleton occurs.

Dr. Wallace Jenson, University of Pittsburgh, made the comment that he agreed with Kehoe's points.

The next subject discussed concerned the ingestion of lead, and this was presented by Dr. Kehoe. I am going to give a very brief summary of Dr. Kehoe's very brief summary of some twenty-five to thirty years of work in lead metabolism.

These were studies of volunteers in which lead, in the form of soluble salts, was added to a diet of which, again, the lead contents were known. The lead metabolism of the subject was followed accurately over some years.

These studies have been extensively reported in the medical literature.

These were the classical types of biological balance experiments in which an accurate measure is kept of food intake and the total output of a substance under study, in this case lead.

When lead was added to the diet at the rate of 0.5 to 2 mg per day, the body burden of lead, as measured by blood lead as an indicator, rose gradually and soon reached a new plateau. At these new elevated levels, a reasonable degree of balance was reached in which no further accumulation of lead occurred in the body.

Kehoe also told us about a clinical observation he made in which two persons, accidentally exposed to 10 to 15 mg of soluble lead by ingestion daily, developed clinical poisoning in a period of about thirty days.

He also told about a human inhalation experiment at 0.15 mg per cubic meter with particle sizes of .05 to .18 micra. In this instance the blood lead increased from a level of from .025 to .030 mg per hundred c.c. of blood to a level of .040 to .050 mg per hundred c.c. of blood.

In another inhalation experiment in which the particulate lead had a mean diameter of 8 micra, the lead was not absorbed by the lungs but, rather, reached the gastro-intestinal tract and was found in the stools.

Kehoe pointed out that an intake of .3 mg per day, or higher, above the present lead intake that "normal" people would have would probably cause an appreciable increase in the body's burden of lead.

At this point Mr. MacKenzie, of the Division of Air Pollution, pointed out that since lead absorption from the respiratory tract was significantly more complete and faster than from the digestive tract, he had made some quick calculations and reached a figure that would indicate that a much smaller quantity than .3 mg would result in a high and rapid accumulation of lead in the body, and so would increase the body burden significantly if it were breathed.

In reply, Kehoe noted that he had made many actual inhalation experiments of human exposures, that his tentative conclusions were that persons exposed to levels of 0.15 mg per cubic meter of lead sesquioxide, a highly soluble material, at particle size of 0.5 micra, for approximately twelve hours per day for six days of each week, would probably--and he said "probably"--reach toxic levels in the body in about two years.

Such inhalation experiments performed, using levels of .10 mg per cubic meter, did not show evidence of increasing body burden to toxic levels.

A question was asked from the floor as to whether there was any information of any changes in human lead burden generally in recent years. Kehoe replied that his data indicated that the lead burden had actually gone down. He said that if this was actually the fact, it might be explained by a reduction in lead that is now being ingested in foods. This reduction of lead in foods, he said, was probably due to less use of lead insecticides and to improved processing and packaging of foods, meaning that the containers in which the foods were prepared and packaged were less likely to have lead in them.

Dr. Cholak of the Kettering Laboratories, the University of Cincinnati, presented data which in a sense confirmed Kehoe's comments. He noted that for a large series of persons studied in Cincinnati both in 1943 and in 1962 there was a remarkable similarity in the findings in these people, for the two studies, in both their urine and their blood lead content.

In a similar vein, Dr. Stokinger, Public Health Service, told us about Leonard Goldwater's studies. Stokinger told us that Goldwater, at a WHO Advisory meeting, had reported on the lead content of urine and blood for a large series of persons, which he had done in fifteen different countries, including three states in this country. Stokinger remarked that Goldwater's findings were that as amongst the countries there was an amazing similarity of the data for the levels of lead for the urine and for the blood over the past two decades.

Stokinger's point in this case, as I understood it, was that Goldwater's data had been collected over a period of some twenty years and there were no differences in Goldwater's findings over that period of time.

A question was asked whether or not anybody had information indicating that lead was useful in any biological system. Kehoe replied that, although lead was not so far found occurring naturally in any known enzyme system, there is an enzyme system about which he knew in which lead was used experimentally to replace a naturally occurring metallic element, and that the system's action was not disturbed. (Goldwater told us about some additional data later on in the second day of the meeting. And I am sure somebody else will tell us about that.)

Kehoe noted that in the existing levels of lead ingestion and inhalation that are occurring in the general population in the United States today "a balance is automatically reached at which there is no increase in body burden to a toxic level."

Dr. Lloyd Tepper's presentation was limited to a discussion of the hazards which might exist from direct skin contact with lead. He divided the subject into three areas: body-retained lead pellets; inorganic lead compounds; and organic lead compounds.

He noted that the body-retained lead pellets are very rarely troublesome as regards their lead content; lead is absorbed very slowly from them, if at all.

In considering the inorganic lead compounds he said that it was extremely unlikely that systemic poisoning occurred from the entry of inorganic lead compounds through the intact skin.

In considering the organic lead compounds, the significant ones were lead tetramethyl and lead tetraethyl.

Dr. Tepper noted that there had not been any cases reported of poisoning from these compounds when they were present up to about 1 percent in concentration in gasoline. Poisoning cases have occurred when they were handled unmixed. They were absorbable to toxic levels through the intact skin.

In experimental animals they were known to cause death even when exposure was limited only to the skin.

The occurrence of human poisoning cases has been rare.

Dr. Kehoe remarked in this connection that, in experimental work with rabbits, tetraethyl of lead was exceedingly poisonous when administered dermally. Tetramethyl of lead was not.

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DISCUSSION

DR. HEIMANN: I would like to take a few moments to make some other comments about the meeting. I want to make these comments based on my listening for the last two days, having some discussions with some people in and outside the room, and on my experience as a physician who has spent most of his life in public health work, mostly in occupational health or air pollution. I have done research in the laboratory. I have done epidemiologic research. I have been an Administrator of Public Health. I have been in the Federal Government. For a while I was assistant to Mr. MacKenzie in the Division of Air Pollution. For a long time I was in the occupational health field. I have been in many places around the world observing what goes on in public health, both for and against the people. I do not mean to get into any acrimonious debate; but I do wish to make some remarks of some conclusions that I reached based on these two days' listening and my background of experience.

I am not intending to impugn anybody's work.

The first thing I'd like to point out is that there has been no evidence that has ever come to my attention, including at this meeting, that a little lead is good for you. I say this because I believe there are some persons who imply that this is so, because of some of the statements which they make.

The second point I would like to make is that it is extremely unusual in medical research that there is only one small group and one place in a country in which research in a specific area of knowledge is exclusively done. There needs to be repetition and extension of the fine work which has been done at the Kettering Laboratory with regard to the metabolism and the balance experiments which have been done on lead. It needs to be repeated in many other places, and be extended.

There needs to be, in my opinion, an extension and improvement of the work which was done by the Public Health Service in its tri-city study of lead body burdens of people in communities. There needs to be a careful study of the figures which I have given as the safe ones at which lead toxicity will probably not occur.

I say specifically that it is our experience in medicine that to use a single figure as the safe one beyond which poisoning will probably occur, and below which poisoning will not occur, is a most unusual kind of a situation in Public Health and in medicine. I believe there needs to be careful study of the figures which are used.

I believe, for example, that a large group of persons whose levels of blood lead ranges from .04 mg per 100 c.c. up to .079 needs to be carefully studied over a long period of time, even for their lifetimes, to determine if any abnormality develops in them which is important to their well-being.

Finally, I want to remind you that there is a natural variation in human reaction to many kinds of environmental factors, and this includes lead.

Therefore, I say, one needs to study this problem a lot more carefully than has been done to determine what are the reasons for this variation in susceptibility. Why do some people become poisoned and others do not?

The final point I wish to make is that there needs to be a thorough evaluation, without political implications or bias, of the studies which have been made in the U.S.S.R.

Do they have information which indicates that lead at the levels that they are talking about causes some abnormality in function which we think is important? Do they or don't they?--regardless of what their politics may be.

MR. CHOLAK: Only a matter of correction of Dr. Heimann's assumption that the figures I gave were for occupational exposures.

The figures for 1942 and those for 1963 were all for unexposed cases; that is, people who were not exposed in industry to lead, unless you include the police and the firemen, who did have some exposure as far as concentrations in the atmosphere is concerned as a result of their standing at very busy traffic intersections.

MR. ROBINSON: One slight correction in the statement as to size distribution of particles.

Seventy-five percent of the mass, according to our studies, is contained in particles less than .42 microns in diameter, assuming for that size calculation a density of 1. This is the Stokes diameter. But 75 percent of the mass of lead is contained by particles of that size.

MR. MACKENZIE: How confident can we be of the density figures that you were citing?

MR. ROBINSON: This is a calculation that if one assumed a lead oxide composition of the particle, or in our determination, which has a density of 6 I believe or thereabouts, one would multiply this size by the square root of 6 to come up with the visible or the microscopic size of the particle. This is a commonly used technique for size distribution for particle size presentation when one is not sure of the composition of the material. In making our size distribution studies, the aerosol spectrometer which we used separates the particles by Stokes settling velocity, by Stokes diameter, and it is more convenient to just assume a hypothetical density of 1, give a diameter of a spherical particle of density 1, that would give you this size distribution.

I think that this is also commonly used by the health physicists in dealing with lung deposition, impaction of particles and such, where the microscopic size is not terribly important; but the aerodynamic properties, the impaction and the retention on these bases are important. So that this is merely one way of getting around dealing with a large number of different densities, that really the parameter which one measures in our case, and which, my understanding is, is important for retention in the respiratory tract, is this aerodynamic size which is expressed normally as a density 1 particle.

DR. HARDY: When Dr. Kehoe was talking of the balance studies, his very elegant studies that represent fundamental knowledge, he talks about "no increase to a toxic level." I think in this statement, the key word is obviously "toxic." And Dr. Kehoe and/or the reporter must say what they mean by "toxic."

DR. KEHOE: I seem to be a bit under the gun, and I've got some difficulties to present here.

I made a very grave mistake in trying to present to this audience, even representing as it does a good many experts, information gathered by very careful, minute attention to detail over a period of twenty or thirty years and then condensing this into twenty minutes of speech.

This gave our reporter, to whom I give the greatest credit for good intentions, some serious difficulties in undertaking to report what I said which was condensed from twenty years of experience and writing. He tried to do this in a matter of a very few minutes and, I am frank to say, with the most generous interpretation of what was said. I didn't recognize what I had said. I am afraid this is a very serious difficulty. Let me illustrate one point which Dr. Hardy just mentioned. I did not say that there was no accumulation in the body as a consequence of the present type of exposure by way of food, water, and air; rather that there was no exposure significant in terms of the toxic levels. I said there was no accumulation.

The balance experiments that were carried out on normal subjects demonstrated quite carefully and completely that within the limits of the experimental error of the observations, intake and output balance. They balance so well that I myself regarded this with a "fishy eye" and, therefore, found it necessary to repeat these experiments time and time again. And what has happened in the case of the normal individual is this: When one follows the normal healthy human subject in the laboratory in balance experiments, according to the techniques that we have carried out, that output is greater than the intake by as much as from 8 to 10 mg per year.

We have interpreted this as meaning that the factor of absorption from the air, which was not measured, was probably the imbalancing factor, and that it amounted to at least as much as the difference between the intake and the output.

And then, when looked at and examined (and this occurred in not one subject but a number, particularly in two experiments in which every item of food taken in was weighed), under these circumstances these differences were so nearly identical in two experimental subjects, as a result of plain arithmetic, that the imbalance was of this order of magnitude: 8 to 10 mg per year.

This amounts to an intake in the air, by this indirect method, of about 20 micrograms per day. And this is the closest approximation that we have in any of the literature at this time as to the amount of lead that is taken in from day to day, and actually absorbed from the air. This is one point that is of extreme importance. I said nothing about toxicity. I carefully said that in this and the other experiments, the criteria of safety are not some dubious figures with respect to toxicity. I carefully said the threat in terms of exposure of ordinary individuals in our society at the present time, the threat that is offered is the evidence of any progressive accumulation which, if it occurs over a month, is insignificant; which, if it occurs over a year, is insignificant; but which may be significant in a lifetime. I'm not talking about a day, a week, a year, I'm talking about a lifetime.

And this is exactly the criterion that we have used in setting the standard (which, incidentally, was used to modify the Public Health Service standard with respect to the drinking water), saying that if you as much as double the present intake of lead in food, which is by all odds the largest feature in what we call the absorption of lead in the normal environment, you are at the point where there is barely some significant increase in the lead concentration in the urine, not in the blood, a difference which is very small, but which you cannot trust over a lifetime.

Now the other thing which enables me to say that there was no accumulation under ordinary circumstances is a series of selected observations on cadavers ranging from three years of age to 97, in which there is not the slighest suggestion of a progressive increase in the quantity of lead in the body, that is, in the concentration of lead in the tissues of the body with age.

Now these simply illustrate the points that I wish to make. If this was misinterpreted by Dr. Heimann, with the best of intentions, it must have been lost on a very considerable number of people in this room. Consequently Dr. Heimann's summary report will have to be reviewed from the point of view of the published data. Everything I've said here has been published, everything can be examined by the critics with respect to whether it is true, whether it is a radical statement, or whether it is a conservative one.

And I submit that in the period of thirty years in which I have been responsible for determining the facts of life with respect to this particular toxic agent, the observations that have been made have been made with an examination which by ourselves, from a point of view of scientific critique, have been more rigid than that which have been applied to any other part of this field.

I suggest, therefore, that in the consideration of this problem the experimental work be examined in detail, that the conclusions of which I spoke briefly here (a number of which have been misinterpreted) be examined in detail, as they must be if you are to interpret the information which is available in this field.

It simply was impossible to present more than fragments of it in the short time that was allotted to me. And it is quite obvious that much of it has been misunderstood.

DR. RADFORD: I'm sure that Dr. Heimann did not mean to imply that the fact that the Kettering Laboratory has had such a prominent place was because of any design. I think it reflects a lack of interest in other laboratories in lead over the years. We would certainly hope that out of a meeting such as this would come greater dispersion of the effort in this field. We look forward to collaboration with other laboratories.

It's obvious that this whole question has become overladen with certain emotional responses on the part of the people involved, and I think this is natural. I don't think we should avoid these. It's perhaps a little unusual that the emotional bias of the individuals involved comes to the surface in a meeting of this kind so readily. Perhaps it's because the trigger, Dr. Patterson's article, is so obviously an emotional article.

I would like to make two main points. First, we aren't concerned only with the problem today. We're obviously thinking ahead thirty years from now. I use the term "thirty years;" it has been used already. It's approximately the time that I have for professional activity. But thirty years or a hundred years, we are concerned with the long-range implications of what may be decided today or next year on future generations.

I became interested in lead in part because of the radio isotope lead-2-10, and I would just like to draw a parallel here between the problem with regard to environmental radiation and the problem of environmental chemicals. I think the history of environmental radiation and the attitudes of the people toward it as a health hazard are very similar to the kinds of things that we are dealing with today, admittedly on an emotional basis.

As you know, in the early days when I first became involved, the idea of a safe exposure was a very prominent one. We were told "if you get .7 rad per week this is safe." And there was a considerable body of opinion that reacted very emotionally at the time that Dr. Linus Pauling and others began to imply that there was no such thing as a safe level of radiation exposure.

We have the feeling now that there is a finite risk for whatever exposure the individual may have, and that we have to balance this risk against the benefits. This is a concept that we now hear a lot about. Similarly, we have to balance the risks of chemical exposure with the benefits they confer. We have heard something about both here.

But I do hope that in the future we look upon this as a distribution, as Dr. Heimann implied. We are not going to find a single answer for any given population group. My feeling is that the possibility of proving no damage is really quite evanescent; that we will never be able to assert there is no risk to any part of the human population. We simply have to do the best we can with the means at our disposal.

Dr. Kehoe was very straightforward, I thought, in emphasizing that as new techniques become available for assessing damage we should use these as our criteria.

I would only say that in the future the Kettering Laboratory will certainly be continuing the work that Dr. Kehoe and many others have carried on so ably over the years. We hope to use better criteria that are becoming available, but were not available in the past.

It has been very stimulating to me to see these points of view come out, because one of the things we talk about in our department is that lead is really a prototype of many other environmental agents, and we are going to have to take exactly the same critical look at each of these as we do with lead.

I think that in effect the whole field of environmental health is, you might say, on trial in terms of defining the risks and benefits that we will derive from use of these materials, so that the public at large can be given a rational basis on which to decide, on political grounds or whatever, that lead should or shouldn't be taken out of gasoline, that pesticides should or shouldn't be used in various situations, that asbestos should be curbed.

MR. MACKENZIE: I think that a couple of items Dr. Radford mentioned have great fundamental importance. There is the matter of establishing so-called threshhold. It has seemed to me for many years that this matter of a threshhold is not so much a definite boundary as it is a range; that physiologic effects (this is what we are concerned with here) do not sharply cut-off at a particular point but, rather, their severity and the type of effect may occur over a broad range of exposure. This may be disputed by some of my friends in pharmacology and toxicology, but this is the way it has appeared to me, that we are not dealing with firm boundaries but, rather, with ranges.

The other point that I would like to comment on is essentially the impossibility of proving a negative. This is something that I think many of our friends do not really grasp effectively. Any experiment or research project that is set up can determine some positive results, and they can go down into ranges where we don't find physiologic effects any more; but if we improve our tools of measurement, we may find some. But any time you come up with an observation in which you didn't find anything correlated with something that you were testing, this, again, doesn't prove anything in my opinion, because you cannot prove the negative: all you can prove is the positive.

DR. KEHOE: I wish to make one additional comment which I think was also rather emphasized by one of Dr. Heimann's statements with reference to the 80 micrograms.

There is no implication that the figure which we have settled upon of 80 micrograms, plus or minus the experimental error of the observation, is any other than just this, the lowest point in the range. And the range which is associated with the occurrence of lead poisoning among people in industry, and, so far as we can see, among people in the general population when they have an adequate exposure, extends all the way through a very wide area.

Let me just illustrate this with one point. It is terribly important in the field of industrial hygiene to have some kind of standard which says "if you will achieve this degree of excellence you will not have cases of lead poisoning."

Over a period of many years, during which we have been following scores of industries with variable degrees of exposure, we have been concerned with finding the lowest level of the concentration of lead, first in the urine. For many years this was too variable to suit my purposes. Or the concentration in the blood.

What we want is the lowest value that is associated with any kind of symptom complex which can be recognized as clinical lead poisoning--and in clinical lead poisoning I and my associates include variations in the hemoglobin synthesis that result in increased porphyrinuria. This, in my estimate, is lead intoxication. When you get abnormal quantities of lead in the urine and in other tissues of the body you are dealing with the first sign, in my experience, of the occurrence of lead intoxication.

The point at which these evidences of lead poisoning cannot be seen, in our experiences, are at the level of 80 micrograms, plus or minus 10; because the error of the method is of the order of magnitude of 10 percent.

This is quite different from saying that there is a point which says lead poisoning. It is not that. This is the point that says no lead poisoning. And there is a great difference in the philosophy of these two situations.

And if I show a little evidence of emotional response, I apologize to this scientific audience. But, on the other hand, it is a very disconcerting and frustrating thing to find that what one has said is distorted by attitudes of mind or in the figures. This is the only reason for my saying that a description of these experiments, which was necessarily presented very hastily, has got to be checked against the books. This will have to be done; because, otherwise, we will be dealing with statements which I didn't make.

MR. SILVER: I am sympathetic with the idea of not making lead a "whipping boy" because of the ease of following it by the chemists while ignoring other toxic factors, the tricresol phosphates, the olefins, the aromatics. The other things that might have to be added if we did away with lead might be more serious than the lead itself.

On the other hand, the kind of toxicology that I was taught in gas engineering at Hopkins (I graduated in 1937) matches Dr. Hardy's and Dr. Heimann's more closely than it has some others.

In engineering we commonly deal with multiple factors that we normally handle by quantifying and summating.

I showed yesterday the X_1, X_2, 1/2, 1/4, and so on, an infinite series: now those factors may be dynamic. If we design a bridge we design for a particular wind: when the wind is not blowing that factor is unimportant; when the wind blows it becomes important.

When you deal with a patient--a physician sends me into a patient's home to try to solve environmental problems for him, I have to look at all the problems. And the patient is not just faced with lead poisoning. I think we have to consider a case that might be 10 percent lead poisoning, 10 percent chlorodane poisoning, 10 percent DDT poisoning, not just lead poisoning. I think we have to look at these types of things.

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SUMMARY: RISK OF EXPOSURE AND ABSORPTION OF LEAD

(This summary is printed in addition to the preceding summary presented by Dr. Harry Heimann, at the request of Dr. Kehoe)

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The next subject discussed was concerned with the question assigned to Robert A. Kehoe, M.D. "Under what circumstances is the ingestion of lead dangerous?" This question was answered at once, in principle, and then in specific terms on the evidence of extensive experimental data, illustrated in brief by the graphic account of two of the experiments on human subjects to whom measured dosages of lead were administered over periods of time ranging from two to four-and-one-half years.

The healthy male adult, who is the ordinary citizen devoid of occupational or other unusual exposure to lead in the U.S., ingests from 0.12 to 0.35 mg. of lead daily with food and beverages, and inhales (by calculation) quantities of the order of 0.010 to 0.045 mg. daily from the ambient air of his community. He eliminates lead in his feces and urine in quantities which, when combined, are equivalent, statistically, to those taken in in food, beverages, and respired air. The evidence of "balance" experiments, conducted over the period beginning in 1939 and continuing at present, indicates that progressive accumulation of lead in the bodies of such persons is negligible over the period of the life span. It is probable, theoretically, that some slight accumulation occurs. It is too small, however, to detect, since it is within the limits of the individual variability occasioned by variation in the dietary intake of lead of the heavy eater as compared to the light.

When, on the contrary, similar experimental subjects are given sufficient quantities of lead, in solution, with their meals (in simulation of experimental contamination of their food and drink), over periods of time sufficient to demonstrate, unmistakably, the course of events, there is a somewhat variable but generally steady rate of increase in the output of lead in the urine, a similar but less rapid increase in the concentration of lead in their blood, and a progressive accumulation of lead in their bodies (cumulative difference between the intake and output of lead as measured day by day) at an essentially constant rate over periods of years (in one experiment over the period of 4.5 years).

The reasonable presumption is that this steady rate of building up the body burden of lead, under the conditions of steady continuous exposure, continues through the lifetime of the individual (since it increases progressively, at an essentially undiminished rate, for so long a period as four-and-one-half years). One concludes, therefore, that the only presently acceptable criterion for safety from the regular ingestion of lead is the limitation of the average quantity ingested daily to a level which will not give evidence of an increase in the body burden of lead over a prolonged period of time. One experiment designed to demonstrate how much lead could be added with safety (from the aspect of no accumulation) to the present level of dietary lead, was carried out over the period of eighteen months (4 weeks or 28 days), during which 0.30 mg. per day was added to that in the diet of a suitable subject. The diet of this subject averaged 0.23 mg. per day, the resultant total intake by ingestion being 0.53 mg. per day. There was a very slight trend (of borderline statistical significance) toward an increase in the concentration of lead in the urine during the experiment; there was no suggestion of a corresponding trend in the concentration in the blood. This relationship between the apparent, slight response in the urine and the lack of response in the blood is in keeping with other observations which have demonstrated the greater sensitivity of the urine to a slight increase in the rate of absorption of lead.

In consequence of these experiments, the threshold point in the regular ingestion of lead by the general population is regarded by investigators in the Kettering Laboratory as being at or near 0.6 mg. of lead per day, under conditions in which the opportunity for the inhalation of lead from the ambient air is not significantly greater than it is at present.

Lead poisoning from ingestion is the consequence of a sufficient intake of absorbable lead by this route, over a sufficient period of time. Evidence, from the study of two cases of lead poisoning that occurred under conditions that could be appraised with reasonable accuracy, has demonstrated that the ingestion of 10 to 15 milligrams of lead per day, under suitable conditions over the period of 1 month, is sufficient to cause poisoning. Much longer periods of time are required to induce similar results at lower levels of dosage. Thus the evidence is that the ingestion of lead at the level of 1 miligram of soluble lead per day (in addition to that in the average diet, approximately 0.30 mg. per day) will reach a dangerous level (not necessarily associated with poisoning, but potentially capable of inducing poisoning) in about 8 years; that of 2 milligrams daily, under comparable conditions, in somewhat less than 4 years, while that of 3 milligrams per day would appear to require only 8 to 12 months.

Later in the session, in response to further discussion and questions concerning the effects of the inhalation of lead, Dr. Kehoe described briefly, and presented some of the results of, a series of experiments in which human subjects, after lengthy preliminary observations on their intake and output of lead under "normal" conditions, were subjected experimentally, within safe limits, to the inhalation of dispersions of known concentrations of known compounds of lead in known states of subdivision (size of particles). (The experiments referred to at this time have been described in detail in the literature, and certain results were illustrated graphically and too briefly to serve other than as partial illustrations.) Certain important and pertinent results were cited:

1. Exposure to air containing lead sesquioxide in a dispersion of particles ranging up to 0.18 micron in diameter (mean diameter 0.05 micron) at the concentration of 0.15 mg. per cubic meter, on the intermittent schedule of 7.5 hours per day on 5 days of each week, results in a prompt increase in the output of lead in the urine and in the concentration of lead in the blood. Within 4 to 6 months, these levels reach their maximum heights, after which, as in the case of occupational exposure that remains reasonably uniform (i.e. without gross increase or decrease in severity), there is no further progressive increase, but only variations above and below the average.

2. The peaks (plateaus) in the levels of concentration of lead in the urine and blood of the subject, under these experimental conditions, occurred at about 0.085 mg. per liter in the case of the urine, and at about 0.042 mg. per 100 grams in the blood. (These levels correspond well with the findings obtained among industrial workmen in an industrial establishment in which they are protected by adequate measures of industrial hygiene against the occurrence of lead intoxication.)

3. Under the conditions of this experiment (respiratory exposure to particles of lead of the mean diameter of 0.05 micron) there is no expectation of upper respiratory deposition of particles, and indeed there was no suggestion of the occurrence of any of the dispersed lead in the feces, whereas, when the lead in the chamber was dispersed in particles of larger size ranging up to 4 microns in diameter (50 percent were smaller than 0.9 microns, 90 percent, smaller than 2) a significant proportion of the lead reached the naso-pharynx as shown by the content of lead in the feces.

4. The retention of lead in the respiratory tract (obviously in the alveoli) during the inhalation of air containing particles of lead sesquioxide ranging up to 0.18 micron in diameter, amounted to 35-40 percent of that inhaled. Absorption from the lung was prompt and apparently complete, as judged by excretory phenomena.

Since intermittent respiratory exposure to absorbable compounds of lead, under experimental conditions, cannot yield results comparable to those associated with continuous exposure (as to the general ambient air), the attempt has been made to derive the results of continuous respiratory exposure by calculation from those obtained under variable conditions with respect to time. Such experiments, both those completed and those in progress, were described briefly. The principle is that of obtaining a series of points representative of the effects of exposure for 3, 6, 9, and 12 hours per day (it being feasible experimentally to engage suitable subjects in such experiments, but not continuously for weeks, months, or years). It appeared, from the preliminary experiments, that it was possible to extrapolate, with reasonable assurance, from such points to the terminal point of 24 hours per day. For example, one derived the conclusion from preliminary experiments that continuous exposure to air containing 0.15 mg. of lead (as sesquioxide) per cubic meter of air, dispersed in particles ranging up to 0.18 micron in diameter, would result in reaching a dangerous level of absorption (indicated by the concentration of 0.08 mg. of lead per 100 grams in the blood) in about 2 years.

Thus far, exposure to air containing 0.010 milligram (10 micrograms) of lead per cubic meter in the same dispersion of particle size as that indicated above (mean diameter 0.05 microns), has not induced any demonstrable increase in the rate of the urinary excretion of lead or in its concentration in the blood, during successive periods of 16 weeks at a time over the total period of about 2 years, within which the individual periods have ranged from 3 hours per day on every other day up to 12.25 hours per day on 6 days of each week (that is, from 10.5 hours per week to 73.5 hours per week). It appears that it will be necessary to increase the concentration of lead in the chamber to a level above 10 micrograms per cubic meter, so as to produce barely positive results in at least three points (in time) from which an extrapolation can be made to the effects of exposure all day on every day.

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SUMMARY: TOXICOLOGY OF LEAD

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The summary prepared for this session by the Chairman and Co-Chairman takes a somewhat different approach than that used in some sessions. It was concluded that, in view of the many comments by many individuals on the principal presentations, it would be impractical to attempt to report the proceedings in a detailed manner. What has, therefore, been attempted is an evaluation of the entire session; inasmuch as such an evaluation involves selection, it necessarily represents personal judgments, and may, therefore, be disagreed with by some of those present.

This particular session consisted of two parts which were in some degree unrelated. The first part was toxicological and dealt with the relationship of lead to health and disease. The second part, and this will be dealt with more briefly than the first, dealt with the present levels of lead in the biosphere and the technological and geological processes by which the present levels were reached.

It seemed to the Chairman and Co-Chairman that there emerged from the first part of the session the sense that there is a considerable body of knowledge of the classical disease of lead poisoning. Furthermore, it appears that there is partial, but an inadequate, understanding of the metabolism and physiological handling of lead at lead burdens associated with classical lead poisoning.

There seems to be general agreement as to the critical features in the diagnosis of classical lead poisoning. In addition, the relationship of body burdens as described by blood and urine levels to classical lead poisoning appears to be reasonably well established. It also seems probable that, with respect to classical lead poisoning, the present thresholds seem acceptable and have been demonstrated to be sound and useful guides to control procedures.

A fair amount is known about lead concentrations in blood and urine in normal populations, including some knowledge of populations in various geographic areas. We know, on the other hand, little about lead burdens in primitive and isolated populations. There appears to be no evidence of an upward trend in blood or urine leads in the U.S. population in the last several decades.

All of this, however, leaves unanswered what is unquestionably the most important issue before this symposium, namely, the relationship of present lead levels to health. Thus, it does not seem possible to exclude the possibility that there may be some contribution to disease of some undescribed sort from either current lead levels of intake or an intake which would be intermediate between current levels and those associated with the threshold of classical lead poisoning.

There are some grounds for supposing that such effects might occur; some of this concern derives from the possibility that there may not be a sharp threshold below which lead is completely harmless.

Nevertheless, no explicit evidence was presented that in any way suggested that present lead burdens were associated with any major contribution to disease in the present U.S. population. This does not exclude the possibility that there may not be some undetected contribution from present lead intakes to the multiple causes of degenerative diseases, cardio-vascular diseases, renal diseases, and neurological disorders.

The practical question that emerges from this is: How can society reach a reasonable level of assurance that an additional increase or even present levels of lead are acceptable or non-acceptable from a health standpoint? The recommendations below suggest ways of attempting to reach some answers to this question.

The second part of the discussion dealt with the question of the present levels of lead in the biosphere.

An interesting and challenging hypothesis has been published by Dr. Patterson which suggests that the present levels of lead found in the biosphere, including the surface soils and the surface waters, have, in large part, arisen as a result of man's technology. That is to say, that the present lead levels would not be present had man not used his ingenuity in mining and in distributing lead in various and sundry forms. That technology has contributed in some degree is apparent to everyone; that it has contributed to the extent suggested in this hypothesis may be correct. However, additional work would seem to be required to nail this down thoroughly. The hypothesis, at the very least, has plausibility and is very interesting. It fully merits a thorough examination.

The following recommendations have, in the view of the Chairman and Co-Chairman, emerged from this session. These recommendations are based on the conviction that society has a right to know whether there is any significant--the word is, critical--contribution from present lead levels to disease. Secondly, although it is important from purely scientific considerations to know how the lead has reached the biosphere, if there is any suggestion that there is a deleterious effect of present lead levels on health, it becomes very important on health grounds to know this in order to aid in determining how to reduce lead exposure.

The recommendations follow:

1. The major need is to define the relevance for health of present typical lead levels and levels intermediate between these and frank lead poisoning. Approaches should include:

   a. Careful study by in-depth prospective epidemiological techniques of exposed groups, and groups receiving more than the normal lead intake but short of that producing frank lead poisoning.
   b. Restudy of previously examined groups as exemplified by the Wenatchee orchard workers.
   c. Continued careful follow-up of children and others with acute poisoning as a guide to the mode of action of lead; recognizing, however, that at these higher lead levels the specific findings may not be directly relevant to nonspecific effects of lead on health.
   d. Extension of studies of the effects on laboratory animals of chronic exposure to low lead levels.
   e. Search for sensitive means for detecting the effect of lead on physiological function and on tissue morphology. Examples would be the use of aminolevulinic acid as evidence of interference in heme synthesis in the one case and mitochondrial changes in the other.

2. Define more accurately the effective absorption of lead by inhalation from typical lead aerosols as found in the ambient air.

3. Develop techniques for better assessment of body burden of lead and the biological mobility of lead in its several biological compartments.

4. Continue and intensify studies on the relative contribution to the biosphere of lead from geological and technological sources. In these studies, major dependence on geochemical techniques will be required. Analysis of biological materials should include measurements of body burdens in isolated populations and in other biological materials separated insofar as practicable from technological contamination.

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DISCUSSION

DR. HARDY: Ninety-nine point three percent meets with my hearty endorsement. I have to protest briefly that there is evidence of lead-related damage not identifiable as classic lead poisoning.

I have listed the following:

Lane's studies on mortality of lead workers; Henderson's Australian work on the relationship between lead and renal disease; Dr. Jenson's work and, more modestly, mine on the asymptomatic lead worker's difficulty with his hematological abnormalities; and then the very large body of work on children without identifiable lead poisoning who, through well documented evidence, have experienced harmful lead effects either in their hematological abnormalities or mental retardation.

I would like to endorse and extend and make a recommendation about the study of the tissues. Since 1963 I have been trying very hard to interest the international biological program which is about to start, in a similar fashion to the international geophysical year, and which includes a worldwide network of laboratories set up to do a number of biological problems, their chief interest seems to be the effect of climate. This has seemed to me to be a unique opportunity to collect tissues from all parts of the world that can be identified as urban, rural, primitive, etc. I haven't been able to find a way, even though Dr. Roger Ravel, who seems to be the American chairman, is congenial to this idea.

One needs some kind of home, and I wonder if Mr. MacKenzie, with all his many other duties, would like to think about this.

MR. MACKENZIE: I might comment briefly on one such proposal that has come to us, Dr. Hardy.

The World Health Organization has been interested in setting up reference centers of various kinds. They have proposed to us, in preliminary form, that a reference center for collection of biological materials and other samples related to factors in the environment (initially in relation to air pollution) might be established.

This is still being discussed.

DR. MARTIN: If I interpreted what I heard in last two days correctly, there seems to be no doubt that more lead is being introduced into humans by food than by any other way.

It would seem to me very fruitful to try and find out what is the lead content of various food products. As I suggested yesterday, in Britain a preliminary survey has been made by taking privets from northern Scotland down to southwest England, and taking these privets in various communities where they are subjected to the coal smoke of Sheffield and Newcastle, to every kind of geological formation from the old red sandstone in southwest England up to the new red sandstone at Larciemouth, and then on this basis to find how much lead there is present in the different vegetation; and then to select various food products and investigate what the range of lead content of the various food products is.

This can be done in a very scientific and unemotional manner, because the techniques and laboratories are available, and we have the geologists, the agriculturalists, and the medical people all combining. It would seem to me that you in the U.S. have a wonderful opportunity to add usefully to your information.

I presented some evidence yesterday to you that even the humble potato may vary in its range of lead content by a factor of 30 to 1. When you do this for potatoes and lettuce, which happen to be the two foods that I have experimented with, you would get most rewarding results.

A lot of this could be done without any great expense and with the facilities that you already have.

I might say that we have done this. We have done quite a good job in Nova Scotia where the Department of Agriculture, the Department of Mines, and the Research Council cooperated in getting soil samples for us which we studied for lead. We found the ones that were high in lead, and then obtained vegetable samples. Strangely enough, we found that some of them had abnormal amounts of lead.

We then went to the physicians and said, "Well, could you tell us anything about these districts, and could we match any of these?" And then because we realized that we were beginners in this, we asked other people who made these lead analyses for us on the vegetables. They found in fact that we did have little areas where there was an abnormally high amount of lead in the vegetation.

This can also be applied by the range people because we have peculiarities, and I am sure you have the same thing in the U.S., where the moose herds in the winter in particular areas completely die out, while a few miles away they will be perfectly well.

We don't know yet whether this is selenium or molybdenum or "what-have-you," but it seems to me that this is the sort of cooperative effort which might be done to our mutual advantage. I would just like to make this plea, because I am quite satisfied that it's in this cooperative effort that we're going to make some big advances.

DR. ROUTH: The subject of this symposium is trying to evaluate how much lead will produce an effect in man. Dr. Hardy has described many of the things that have been alleged to be the effects of lead poisoning.

Two of her subjects this morning were Dr. Jensen's cases, which were people with gross overexposure to lead. If you look at their blood lead, they were markedly in the level above what Dr. Kehoe had been talking about. So this isn't the kind of thing we are discussing today.

Now as far as Dr. Lane's studies, I made a trip to Europe to ask him this question. And the kind of exposure that these people have (cardiovascular effects) were people with 200 micrograms and probably 300 micrograms per liter of urine.

DR. HARDY: The point I was making was not about the indices that you are referring to, but that they were not sick and didn't have classic lead poisoning. The exception that I took to my friend, Dr. Nelson, was that he said the evidence was not available that there was lead damage below classic lead poisoning. You're quite right, these people weren't sick, and this is a key point. Because Dr. Kehoe is the man who set the stage quite correctly in saying that the doctor can identify classic lead poisoning. These people did not have classic lead poisoning, even though they were inhaling amounts of lead above the so-called safe limit. This I think is very important.

So you and I are sort of saying the same things in different ways. But I am the clinical fellow protesting that there is lead damage even though the man isn't ill.

DR. KEHOE: This point of when a man is ill and when he is not ill is, of course, one of the greatest problems with which we are confronted in industry. And I am completely confident that we would have wiped out the greatest portion of lead poisoning, not only in the U.S. but the rest of the world, if it were not for the fact that there is exposure of a serious sort that does not result in illness. And this is in a different area.

In industry the incidence of lead poisoning varies with the severity of exposure, but only on a statistical basis. It is not unexceptional (it is virtually the rule) that as you go into an industrial population that is heavily exposed to lead, you will find from time to time cases of poisoning, and even from time to time cases of wrist drop and encephalopathy. And working beside this man is a man who has as much lead burden as he has, but who has no evidence of classical lead poisoning.

Here is distinctly the area in which we need to know whether there are insidious effects; whether this man is as well as he thinks he is; whether this man's ability to work is an expression of wellness from the point of view of lead.

This kind of information we very badly need, this is the area in which we need the best clinical knowledge, judgment, and scientific investigation that we can possibly find. This is the important aspect in our area of occupational medicine, with respect to lead.

And this, I submit, may be related to the problem that we're talking about, namely, exposure to the lower levels. It may be, it is possible; it is not probable.

You'll have to take my word for that, if this is an arbitrary statement; and it is. But this is based on many years of experience with this particular problem.

What we are concerned with is the fact that there is no lead burden which, at the present time, can be associated with any degree of illness or abnormality. I have seen individuals at the level of .65 in the blood in whom we were quite unable to find any evidence of illness. This is almost incredible, and it indicates that there is a factor, which we do not at present understand, that converts a body's burden of lead, which is innocuous today, or apparently so, to an acute intoxication tomorrow or the next day.

And this is the thing that I want to know, that we've been trying to find out. This is the place where we need the assistance of the best metabolic biochemists that our society affords, because here is a real problem: What is the mechanism of lead poisoning? What is it that takes a quantity of lead in the body which produces no symptomatic result today and converts it into a sick man tomorrow? This is something we do not know, something we very much need to know.

I do not believe that this occurs in a situation to which we as an entire population have been exposed for thirty years. We do not see anything that we can identify as injury in the individual who is exposed to the ordinary situation, minus extraordinary situations in the community, minus occupational exposure. We do not see illness at this point. The physicians in the U.S. and the rest of the world can't all be wrong. They don't find lead poisoning in these people, except in association with a significant degree of exposure above the level about which we are talking.

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SUMMARY: CONTROL OF LEAD POLLUTION

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The session was opened by a very comprehensive discussion of the possibilities of removal of sources of lead contamination in the environment by Dr. Melvin W. First, Harvard University. The speaker opened his discussion by stating that we must first determine whether a reduction in existing lead levels is desirable and necessary. If we conclude that a reduction is necessary, then we must decide how best to do this.

The speaker stated that there appeared to be three major sources of lead contamination of the environment. These are contaminations from: (1) naturally occurring lead; (2) industrial effluents; and (3) consumptive uses. Of these, only the latter two seem susceptible to any significant amount of control.

There is insufficient information on the contribution which industrial effluents make to the contamination of land, water, and air. It is important that this information be obtained. The consequences of industrial contaminations are usually local in nature because the effluent tends to remain close to the source of emission. The application of controls now exercised in this area is much less than would be the case if the objective was to reduce lead emissions to the lowest possible point. This is because little has been done to control lead emissions except for in-plant health hazards, and because control beyond this point has not been justifiable on the basis of economic considerations alone.

Some environmental lead contamination is directly attributable to the widespread use of lead or lead-containing products, because no other substance or group of substances can produce equivalent results as cheaply, either by reason of lead's chemical and physical properties or its relatively great abundance. As an example it was pointed out that storage batteries could be made without lead, but for practical purposes these cannot be substituted for lead batteries because of cost and inadequate supplies of the necessary materials. This example illustrates the dilemmas encountered in attempting to eliminate much of the lead in our immediate environment. On the other hand, the lead content of food and water might be significantly reduced by prohibiting: (1) the unnecessary use of lead in food processing and storage equipment; (2) the use of lead in pipes; (3) the growing of crops near major sources of lead.

A reduction of consumptive lead contamination could be achieved by prohibiting the use of lead in gasoline. This approach is simple in concept but very complex in application. Two possible alternatives, the substitution of diesel or turbine engines for the conventional gasoline engines, and the use of filters to screen out lead effluents from existing engines, are not attractive at this time. Before recommending to eliminate lead from gasoline, we should be sure that it is necessary to do so, and this will require information not presently available.

The question of the use or non-use of lead in gasoline received further detailed consideration from a panel of speakers. Representatives of three industrial firms agreed that it is theoretically possible to produce non-leaded gasolines meeting octane ratings of current leaded gasolines, but at a price. The consumer would have to pay more for such gasoline, although estimates of how much more differed. The production of lead-free gasoline would require a greater consumption of crude oil per gallon of finished product, and a considerable capital investment in new refinery equipment. The economics would favor large producers as against small producers and might result in the elimination of many of the latter. The discussers presented different estimates of the incremental cost of lead-free gasoline at the refinery, the estimates ranging from approximately one to approximately three cents per gallon. Differences likewise existed in estimates of the capital expenditures required for the conversion of existing refineries to lead-free operation, the estimates ranging from 1.5 to 3 billion dollars. The current annual expenditure for refinery modernization and expansion is 500 million dollars, and presumably this expenditure would have to continue in any event.

Following the presentation concerning the possibility of non-leaded gasolines, Dr. Patrick Barry commented on some of the observations being made in England and Europe on environmental lead values and current body burdens of lead. These are, in general, similar to those observed in the U.S. Dr. T. J. Chow presented information on methods being used for the determination of lead isotope ratios in geochemical studies. He indicated that these ratios may be useful in identifying sources of environmental lead contaminations.

In general discussion, questions were raised on the nature and quantity of emissions from engines burning lead-free gasoline. For example, would there be any significant increase in poly-nuclear aromatics or other hydrocarbons of toxicological significance, or in smog-forming components? Questions were also raised concerning the operability characteristics of vehicles using lead-free gasoline.

It was apparent that the answers to a great many relevant questions are not yet available. Before a sound decision can be made on the necessity for and feasibility of a change to lead-free gasoline, it is imperative that more information be obtained on the economics, the health implications, and the potential for smog formation that relate to the change.

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DISCUSSION

MR. GERHOLD: Quite a lot has been said with regard to the failure of many refiners to survive the kind of change with regard to leading practices that has been contemplated; I would simply like to warn this group that there have been similar predictions of failures of small refiners on practically every processing change that has ever taken place, and by and large, these have not materialized. They have not materialized to a very considerable extent, we in Universal Oil are proud to say, because of our success in adapting processing techniques to the special needs of these companies. There is quite a lot that can be done with regard to the adjustment of processing methods to particularly fit the economics of the small versus the large refinery. A change of this kind takes place--if it takes place-over a period of a number of years. There is a lot of opportunity for ingenious adaptation of and the creation of new technology to fit these problems.

DR. HARDY: I was particularly pleased with the last speaker because in my 20-odd years of work in the field of man-made disease I have been enormously impressed in my lay fashion with the versatility and speed with which industry can accommodate itself to demands legitimately made.

Now I'm sure you'd think I was letting the side down if I didn't bring up Dr. First's point, that the economics of the situation demand that lots of people are hurt and become sick before anybody brings up the question of too much lead in the air.

Now that isn't what either Dr. First intended or what you intended, but it does come through to the listener that the dollar sign is terribly important. And with the wisdom and cleverness of American industry and all the interesting things that we can find to do, I would hate to see us end on that note.

The other pot-shot I'd like to take at my Boston colleague is his bringing up the reaction to the Surgeon General's report on cigarette smoking. The cigarette is a self-imposed risk. If a man has put before him what the relative risks are, it seems to me he can make his own decision, and should be allowed to make his own decision. This is a very different matter from the deposition of lead on soil that can then get into a food chain or into water supplies or conceivably is eaten directly by children.

So I'm with Dr. Nelson and others. Let's get things sort of in perspective.

MR. GAMMELGARD: I was deadly serious this morning when I said that if lead-free gasoline is imposed on the refiners of this country, there will be dozens of refiners who will go out of business the morning after that regulation becomes effective.

I would like to point out that Universal Oil Products does not operate refinery number 1, and I think that the advice coming from people who do operate refineries, as far as refinery economics are concerned, might be a little more acceptable than from someone whose primary interest is in building refinery equipment, and designing it.

I have absolutely no bone to pick with the integrity or the competence of the Universal Oil Products Company; this is not my point. I will admit that you did a fine job in bringing out fluid cat crackers for the small plants. There are a number of small plants, as we all know, in this country that do not have cat cracking. There are still more than do not have alkylation.

To do this lead-free job at the current quality levels will require 100-plus octane permium, 94 octane house brand. I do not see how a small refiner could do this without hydro-cracking, and I don't think you do, without additional cat reforming, and without cat cracking, without alkylation.

I can produce a number of refiners. I happen to be president of the National Petroleum Refiners Association. We have the largest companies in the country and some of the very smallest. I have heard a number of these men from the smaller refineries express an extreme concern as to how they would be able to raise the money to build these expensive additions that would be required for lead-free gasoline.

MR. MACKENZIE: I think the economics is of importance and interest, but the overriding consideration is the effect on health. This is what we must look at primarily.

Most of the material that has been submitted relates to how much it's going to cost the industry and this can be translated by arithmetic into what it costs the average consumer.

The average consumer drives a car, according to the best statistics that I can recall, a little less than 10,000 miles a year. If you translate this into gasoline costs, this may mean an additional cost, if we're talking about 1 to 3 cents as was mentioned here, something in the range of \$7 to \$21. How this relates to the total cost of his operating the car for 10,000 miles over the period of a year I will leave to your mental arithmetic or arithmetic on paper.

One further item, however, that I think we have not brought out adequately is that there are other advantages to the consumer in relation to these costs that he would have to bear and these are concerned with undefined items relative to automobile maintenance, the longevity of the engine, and so on.

These have been talked about but really good numbers have not been put on them. They, however, should be kept in mind.

There is a further point that I think needs to be considered. The lead has been shown to be a poison in the catalyst if we use such an approach for air pollution control of the exhaust from vehicles. Now, at our present stage of development of technology, we are not using catalysts. The automobile manufacturers are not using catalysts for the control of pollutant emissions from the automotive vehicles. They do not consider it to be necessary; they can accomplish adherence to such standards as have been promulgated by other mechanisms.

However, inevitably the population of automobiles in this country is going to increase. Whether in the future we are going to have to extend our technology to other procedures than those which are currently being incorporated in automobile design remains to me an open question. I think we need to continue to look at this as we proceed.

DR. FIRST: I'm sorry if the implication came through that the dollar sign was important; I thank you for crediting me with good intentions. I would say more about it, except that Mr. MacKenzie has stated a position which coincides exactly with mine.

I do not feel that the dollar sign is all important, but I do feel that if we are going to cause a large expense to people, we should be in a position where we can justify it to ourselves and to them.

Now I'd like to speak a little more on the second point that you brought out. This was the matter of cigarette smoking.

I agree with you of course, whether one smokes or doesn't smoke is an individual decision and in an assemblage such as ours, with people so much concerned with contamination of the atmosphere, we still find quite a few people smoking. So obviously these people have added up the risk and the benefits and they have made their own personal conclusion.

I am only trying to suggest that when we talk about a universal effect such as the removal of lead from gasoline which might--and this we don't know yet--cost a perceptably higher number of dollars per year for everyone in the U.S., this becomes a political decision which we all participate in, and we may react in the manner in which we have to cigarette smoking.

With regard to the situation in California concerning smog control devices--I am sure that people who have been intimately following it have been aware of the fact that there hasn't been universal gleeful acceptance of this situation of adding expensive things to automobiles by the general public. Far from it. As long as it was a political decision, you do something, everybody was for it; but now that everyone has to spend very large numbers of dollars, and will spend more in the future, I think the enthusiasm among the general electorate may be cooling slightly.

DR. TOMASHEFSKI: I realize that I am far from central; in fact I am probably the most extremely peripheral spokesman here as far as the make-up of this group is concerned, but I should feel very unhappy to have this meeting come to a close without reflecting in the summary of the control the approach which we might take to secure the minimization of the effect of this problem. I touched on this briefly the other day.

But I think that we might think in terms of examining the contribution which the local or the state health departments, or the Public Health Service in divisions other than the Division of Air Pollution which has the specific mandate, and also the contribution of industry, to setting up the availability of whatever screening devices seem appropriate.

I would suggest the possibility of free blood and urine lead analyses and a variety of other services, so that these cases could be detected earlier, and could be detected without the encumbrance of the economic consideration of "Who is going to pay the $10 or $15 that it costs to run the test?" Therefore, every person, primarily every child but adults also, in the community could be examined if there was some basis for suspicion, or could be examined even more. This could be done more broadly if you wished to do a screening process but at least to catch them early with some kind of an acceptable index.

If you are interested in a description of a control effort in one of the larger cities, specifically Philadelphia, I would refer you to an article in Public Health Reports, I have forgotten which year--1963 or 1964--in which the reflection of a number of community agencies, individuals, and industry, were pooled in order to pick up these cases early and secure treatment and reporting.

This might help in a number of ways, but the way in which I'm most concerned is that it might prevent a lot of these youngsters who are sitting on a powder keg of elevated blood lead levels from going on to the point of the explosion of self-destruction.

MR. NELSON: I merely want to ask a question in regard to this problem of traffic and its contribution to lead in the air.

Isn't the concentration of lead in the air from this source a matter of traffic density? And have not we reached, perhaps in at least our major cities, the limit to that traffic density, so that even though more automobiles may get on the roads in the future, we are not going to have perhaps more automobiles per square mile in our major cities, and therefore the theoretical concentrations of lead should perhaps not rise.

The second comment I want to make was in regard to the previous speaker who suggested providing free analytical services. I would merely mention that lead analyses which have usually been long, expensive, laborious, have now been reduced very greatly in cost and time by means of atomic absorption so that a urinalysis of satisfactory accuracy can now be done in a matter of a minute or two and at a very, very low cost.

MR. MACKENZIE: I want to comment on one of Mr. Nelson's comments with respect to vehicle density. It has appeared to us that the more significant parameter to which lead in the atmosphere might be related is the number of vehicle miles travelled within a specific area because this relates to the gasoline consumption and, therefore, the exhaust emission.

I don't yet personally see any limit that is being reached on this because of the continuing construction of throughways, freeways, and highways generally in urban areas.

Certainly this is a trend in most of the cities that I am acquainted with and I suspect that the gasoline consumption per square mile may still be going up.

MR. SILVER: In aircraft engineering we used to quantify what we called the weight cost. There is a problem here of quantifying the health costs.

Now I have been attempting to do this with a few charts which are still full of tea leaves rather than accurate, but if anyone wants a set of these, if you'll write to me, I'll be glad to send you a set of them.

DR. HORTON: Taking up one of Dr. Heimann's suggestions, I would like to make a few non-political comments on the Russian air standards, just to show it can be done. These have been referred to indirectly several times, and Dr. Patterson has also referred to them.

The first comment that needs to be made has been made once before--and I will repeat it in somewhat different words--is that the Russian point of view in establishing standards is quite different from ours.

If you have heard Professor Ryazanov speak on this or read some of his writings, you will have seen that the Russians consider any demonstrable change as undesirable. The standards are used not in the sense that we would ordinarily use them, as something to adhere to, but are propounded as ideals to be eventually achieved, if possible. I think this must be kept clearly in mind when one is considering anything about these standards.

We in this country do not necessarily agree that any demonstrable change is undesirable. I think that this is a position rather difficult to defend, and I notice in the Expert Committee Report of the World Health Organization on this subject, the Russian standards are listed in the first level which is stated to not be affecting health. Professor Ryazanov is a member of this Expert Committee and I presume agrees with this.

There is not one Russian air standard for lead--there are at least three. One is for industrial areas and is based on a 36-hour work week. One is for ambient air exposure. Both of these are concerned with presumably soluble lead compounds. There is a separate standard of occupational work, for compounds like lead sulphide which is insoluble.

This work is published, as are all the standards, in books which come out periodically. There are seven of these books so far published. The Public Health Service has arranged by means of a grant to have these translated.

The first lead standards are to be found in book 1. These are calculated standards without supporting experimental data specifically designed for the purpose. There are also comments on the lead standards in books 4 and 5. Dr. Patterson referred to those in book 4.

The present supporting evidence for the occupational standard which can be found in book 4 is a group of experiments on rabbits exposed to different lead levels by inhalation, plus a control group. Two experimental levels of exposure were used. The effect measured is that of alteration in the coproporphyrin excretion of the urine. It's a quantitative measuring system.

In the fifth book you will find that the present standards for lead in the community air are based on two observations in groups of children living under different exposure conditions. These standards are based on two things: One, that there are differences in the urine lead level in the two groups of children observed; and that there are differences in the coproporphyrin excretion in the urine in these groups of children.

Former attempts to set standards for lead on other bases, such as the one the Russians frequently use of altered conditioned reflexes or altered natural reflexes, are not now used in support of the lead standards.

I would like to emphasize that both the occupational standard and the present one for ambient air are based on single sets of observations; that is, there is not extensive information supporting these at the present time. In using two different groups of children, this work does not take account of the fact that they may differ in other respects than those which are specifically measured. That is, there is no attempt made to measure dietary lead differences or any other lead exposure differences in this case. I think it is essential from our point of view that further work would need to be done in more detail, under different circumstances, with better definition of other possible lead exposure risks.

The use of coproporphyrin excretion in this case is of interest, as are other concepts for determining the risks of various exposures which the Russians employ. But the very limited experimental and observational evidence with which the Russians appear to be satisfied seems to me to be totally inadequate.

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